Dwg.0/0

L21 ANSWER 38 OF 44 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 5
AN 1998-312176 [27] WPIDS
DNC C1998-096289
TI Treating or preventing diseases mediated by TNF-alpha - by co-administration of **antagonists** of TNF-alpha and **IL-12**, having synergistic effect in cases of e.g. **rheumatoid arthritis**, Crohn's disease and transplant disease.
DC B04 D16
IN BRENNAN, F M; BUTLER, D M; FELDMANN, M; MAINI, R N; MALFAIT, A A M
PA (KENN-N) KENNEDY INST RHEUMATOLOGY
CYC 80
PI WO 9822137 A1 19980528 (199827)* EN 64p
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9749599 A 19980610 (199843)
EP 936923 A1 19990825 (199939) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
ADT WO 9822137 A1 WO 1997-GB3151 19971117; AU 9749599 A AU 1997-49599 19971117; EP 936923 A1 EP 1997-912367 19971117, WO 1997-GB3151 19971117
FDT AU 9749599 A Based on WO 9822137; EP 936923 A1 Based on WO 9822137
PRAI US 1996-749979 19961115
AB WO 9822137 A UPAB: 19980709
Method for treating or preventing a disease mediated by TNF alpha by co-administration of a TNF alpha **antagonist** (I) and an **IL-12 antagonist** (II).
USE - The method is used to treat (or prevent recurrence of) autoimmune, chronic or acute immune, inflammatory or neurodegenerative diseases, specifically **rheumatoid arthritis**, Crohn's disease and diseases associated with transplantation (of kidney, heart, marrow, liver, pancreas, small intestine, skin and lung,)infections, TNF-secreting cancers, cachexia and alcohol-induced, or other forms of, hepatitis (claimed).
ADVANTAGE - When used together, (I) and (II) provide a rapid and sustained alleviation of TNF-mediated disease, with significantly better response than when either component is used alone. This permits doses, and thus costs and side-effects, e.g. allergic responses, to be reduced.
Dwg.2A/7

L21 ANSWER 39 OF 44 USPATFULL
AN 1998:161997 USPATFULL
TI **Antibody** to interleukin-12 receptor
IN Gately, Maurice Kent, Pine Brook, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
Wu, Chang-you, Belleville, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5853721 19981229
AI US 1995-381059 19950131 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Feisee, Lila; Assistant Examiner: Sun-Hoffman, Lin
LREP Johnston, George W., Tramaloni, Dennis P., Kass, Alan P.
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 33 Drawing Figure(s); 22 Drawing Page(s)
LN.CNT 1418
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

TI Methods and compositions for modulating responsiveness to corticosteroids
IN Sekut, Les, Westborough, MA, United States
Carter, Adam, Newburyport, MA, United States
Ghayur, Tariq, Grafton, MA, United States
Banerjee, Subhashis, Shrewsbury, MA, United States
Tracey, Daniel E., Harvard, MA, United States
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)
PI US 6054487 20000425
AI US 1997-820692 19970318 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Lahive & Cockfield, LLP
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject. In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) **antagonist**. In another embodiment, the agent is an interleukin-12 (**IL-12**) **antagonist**. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment, the agent is an anti-**IL-12** monoclonal **antibody**. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the treatment of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable carrier.

L19 ANSWER 13 OF 18 USPATFULL
AN 1999:155952 USPATFULL
TI Dihomo-seco-cholestanes
IN Barbier, Pierre, Rixheim, France
Mohr, Peter, Basel, Switzerland
Muller, Marc, Saint-Louis, France
Self, Christopher, West Caldwell, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5994569 19991130
AI US 1998-115188 19980714 (9)
PRAI EP 1997-112225 19970717
DT Utility
FS Granted
EXNAM Primary Examiner: Dees, Jose' G.; Assistant Examiner: Badio, Barbara
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1220
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polyunsaturated 24a,24b-dihomo-9,10-secocholestane derivatives of

condition, tissue specific autoimmunity, degenerative autoimmunity, **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

IL-12 p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L19 ANSWER 3 OF 18 WPIDS (C) 2002 THOMSON DERWENT
AN 1999-458684 [38] WPIDS
DNC C1999-134705
TI New **antibodies** to human interleukin-12, used for treating diseases associated with increased **IL-12** bioactivity such as autoimmune disorders, e.g. multiple sclerosis.
DC B04 D16
IN GATELY, M K; PRESKY, D H; GATELY, M
PA (HOFF) HOFFMANN LA ROCHE & CO AG F; (HOFF) HOFFMANN LA ROCHE INC
CYC 85
PI WO 9937682 A2 19990729 (199938)* EN 46p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
ZA 9900452 A 19990929 (199947) 48p
AU 9925177 A 19990809 (200001)
BR 9907743 A 20001017 (200056)
EP 1049717 A2 20001108 (200062) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU NL PT SE
US 6225117 B1 20010501 (200126)
CN 1288468 A 20010321 (200137)
KR 2001034315 A 20010425 (200164)
MX 2000007124 A1 20010301 (200170)
JP 2002501085 W 20020115 (200207) 50p
ADT WO 9937682 A2 WO 1999-EP202 19990115; ZA 9900452 A ZA 1999-452 19990121; AU 9925177 A AU 1999-25177 19990115; BR 9907743 A BR 1999-7743 19990115, WO 1999-EP202 19990115; EP 1049717 A2 EP 1999-904780 19990115, WO 1999-EP202 19990115; US 6225117 B1 Provisional US 1998-72333P 19980123, US 1999-232522 19990119; CN 1288468 A CN 1999-802310 19990115; KR 2001034315 A KR 2000-708036 20000722; MX 2000007124 A1 MX 2000-7124 20000720; JP 2002501085 W WO 1999-EP202 19990115, JP 2000-528602 19990115
FDT AU 9925177 A Based on WO 9937682; BR 9907743 A Based on WO 9937682; EP 1049717 A2 Based on WO 9937682; JP 2002501085 W Based on WO 9937682
PRAI US 1998-72333P 19980123; US 1999-232522 19990119
AB WO 9937682 A UPAB: 19991122
NOVELTY - New **antibodies** to human interleukin-12 are produced using a mammal which is deficient in the gene encoding the p35 or **p40** subunit of **IL-12**.

DETAILED DESCRIPTION - (A) An **antibody** to the human interleukin (**IL**)-**12** p75 heterodimer which consists of a p35 subunit and a **p40** subunit, where the **antibody**:

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with an **epitope** presented by the **p40** subunit; and

(ii) is produced from a mammal, preferably a mouse which is deficient

in the gene encoding the p35 subunit or the **p40** subunit of **IL-12**.

INDEPENDENT CLAIMS are also included for the following:

(1) a monoclonal **antibody** (MAb) to human **IL-12** which consists of a p35 subunit and a **p40** subunit forming a p75 heterodimer, where the MAb;

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with any **epitope** presented by the **p40** subunit; and

(ii) neutralizes at least 90% of the bioactivity of human **IL-12**;

(2) a hybridoma that produces an **antibody** as in (A) or (1).

ACTIVITY - The **antibodies** can neutralize **IL-12** bioactivity as determined by ability to block **IL-12** stimulated phytohemagglutinin A (PHA)-activated lymphoblast proliferation and interferon- gamma production by PHA-activated lymphoblasts. The 5F2, 16F2, 16G2 and 20E11 **antibodies** were able to inhibit human **IL-12** stimulated PHA activated human lymphoblast proliferation by at least 90%. These anti-human heterodimer specific **IL-12 antibodies** were able to inhibit greater than 90% of **IL-12** stimulated IFN-gamma production when used at 0.5 micro g/ml.

USE - The **antibodies** can be used for controlling diseases with pathologies that are mediated through immune mechanisms, particularly diseases associated with increased **IL-12** bioactivity that results in aberrant Th1-type helper cell activity like autoimmune disorders, e.g. multiple sclerosis, **rheumatoid arthritis**, autoimmune diabetes mellitus, and inflammatory bowel disease (IBD) including Crohn's disease and ulcerative colitis (claimed). They can also be used to treat transplantation/graft-versus-host disease and septic shock.

ADVANTAGE - The anti-**IL-12 antibodies** exhibit higher potency and greater efficacy than known heterodimer specific **IL-12 antibodies**.

Dwg.0/7

L19 ANSWER 4 OF 18 USPATFULL
AN 2002:157653 USPATFULL
TI Triazine compounds
IN Ono, M, Lexington, MA, UNITED STATES
Sun, Lijun, Harvard, MA, UNITED STATES
Zhang, Shijie, Nashua, NH, UNITED STATES
Przewloka, Teresa, Burlington, MA, UNITED STATES
James, David A., Cambridge, MA, UNITED STATES
Ding, Wenli, Worcester, MA, UNITED STATES
Wada, Yumiko, Waltham, MA, UNITED STATES
PI US 2002082259 A1 20020627
AI US 2001-6624 A1 20011130 (10)
RLI Continuation-in-part of Ser. No. US 2000-594362, filed on 15 Jun 2000, PENDING
DT Utility
FS APPLICATION
LREP Y. ROCKY TSAO, Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
CLMN Number of Claims: 48
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 879
AB This invention relates to triazine compounds of formula (I): ##STR1##

R.sub.1 is , aryl, ##STR2##

01/02

9/512701

9/512930

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NEWS 15 Dec 10 DGENE BLAST Homology Search
NEWS 16 Dec 17 WELDASEARCH now available on STN
NEWS 17 Dec 17 STANDARDS now available on STN
NEWS 18 Dec 17 New fields for DPCI
NEWS 19 Dec 19 CAS Roles modified
NEWS 20 Dec 19 1907-1946 data and page images added to CA and CAplus

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=> e leonard john p/au

E1	78	LEONARD JOHN M/AU
E2	3	LEONARD JOHN N/AU

```
E3           88 --> LEONARD JOHN P/AU
E4            1     LEONARD JOHN PATRICK/AU
E5            1     LEONARD JOHN PAUL/AU
E6            2     LEONARD JOHN R/AU
E7            3     LEONARD JOHN S/AU
E8            1     LEONARD JOHN T/AU
E9            1     LEONARD JOHN W JR/AU
E10           1     LEONARD JOHNATAN N/AU
E11           1     LEONARD JOHNSON F/AU
E12           4     LEONARD JON/AU

=> s e3-e5

L1           90 ("LEONARD JOHN P"/AU OR "LEONARD JOHN PATRICK"/AU OR
"LEONARD
                JOHN PAUL"/AU)

=> e leonard j p/au

E1            1     LEONARD J M R M/AU
E2          208     LEONARD J N/AU
E3          346 --> LEONARD J P/AU
E4            4     LEONARD J P */AU
E5           88     LEONARD J R/AU
E6            3     LEONARD J R 3RD/AU
E7            3     LEONARD J R III/AU
E8           30     LEONARD J S/AU
E9            2     LEONARD J S JR/AU
E10          43     LEONARD J T/AU
E11         688     LEONARD J V/AU
E12          45     LEONARD J W/AU

=> s e3-e4

L2          350 ("LEONARD J P"/AU OR "LEONARD J P *"/AU)

=> e goldman samuel/au

E1            2     GOLDMAN S Z/AU
E2            1     GOLDMAN SAM/AU
E3           12 --> GOLDMAN SAMUEL/AU
E4            6     GOLDMAN SAMUEL C/AU
E5            1     GOLDMAN SAMUEL D/AU
E6           50     GOLDMAN SAMUEL J/AU
E7            1     GOLDMAN SAMUEL JAY/AU
E8            1     GOLDMAN SAMUEL L/AU
E9            5     GOLDMAN SAMUEL M/AU
E10           1     GOLDMAN SANDY/AU
E11           6     GOLDMAN SARAH/AU
E12           2     GOLDMAN SARAH A/AU

=> s e1-e9

L3           79 ("GOLDMAN S Z"/AU OR "GOLDMAN SAM"/AU OR "GOLDMAN
SAMUEL"/AU OR
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"GOLDMAN SAMUE
                L J"/AU OR "GOLDMAN SAMUEL JAY"/AU OR "GOLDMAN SAMUEL
L"/AU OR
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                "GOLDMAN SAMUEL M"/AU)

=> e goldman s/au

E1            3      GOLDMAN RUTH/AU
E2            1      GOLDMAN RUTH E/AU
E3         1413 --> GOLDMAN S/AU
E4          237      GOLDMAN S A/AU
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E7           19      GOLDMAN S C/AU
E8            2      GOLDMAN S D/AU
E9           17      GOLDMAN S E/AU
E10          15      GOLDMAN S F/AU
E11           9      GOLDMAN S G/AU
E12          11      GOLDMAN S H/AU

=> s e3

L4         1413 "GOLDMAN S"/AU

=> e o'hara richard/au

MISMATCHED QUOTE IN EXPAND TERM
MISMATCHED QUOTE IN EXPAND TERM
MISMATCHED QUOTE IN EXPAND TERM
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MISMATCHED QUOTE IN EXPAND TERM
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off or masking.

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E2            1      OHARA RETUKO/AU
E3            0 --> OHARA RICHARD/AU
E4            3      OHARA RIE/AU
E5            5      OHARA RIEKO/AU
E6          101      OHARA RIICHIRO/AU
E7            1      OHARA RIKURO/AU
E8            2      OHARA RISA/AU
E9            2      OHARA RUMIKO/AU
E10           3      OHARA RURI/AU
E11           4      OHARA RURIKO/AU
E12           4      OHARA RYO/AU

=> e o hara richard/au

E1            3      O HARA RAYMOND J/AU
E2            1      O HARA RAYMOND K/AU
E3            1 --> O HARA RICHARD/AU
E4            1      O HARA RICHARD J/AU
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E7           21      O HARA RICHARD M JR/AU
E8            2      O HARA ROBERT/AU
E9            2      O HARA ROBERT B/AU
E10           1      O HARA ROBERT D/AU
E11          26      O HARA ROBERT J/AU
E12           1      O HARA ROBERT M/AU

=> s e3-e7

L5           25 ("O HARA RICHARD"/AU OR "O HARA RICHARD J"/AU OR "O
HARA RICHARD
                JR"/AU OR "O HARA RICHARD K"/AU OR "O HARA RICHARD M
JR"/AU)

=> e o hara r/au

E1            1      O HARA PHILLIP M/AU
E2            5      O HARA PHYLLIS L/AU
E3           78 --> O HARA R/AU
E4           15      O HARA R B/AU
E5            8      O HARA R C/AU
E6            2      O HARA R D/AU
E7            2      O HARA R E/AU
E8           50      O HARA R J/AU
E9           32      O HARA R K/AU
E10           7      O HARA R M/AU
E11          48      O HARA R M JR/AU
E12           1      O HARA R P/AU

=> s e3

L6           78 "O HARA R"/AU

=> s e11

L7           48 "O HARA R M JR"/AU

=> s l1-17

L8         2069 (L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7)

=> s l8 and arthritis

L9           23 L8 AND ARTHRITIS

=>  s l9 and il-12

L10           4 L9 AND IL-12

=> d bib ab 1-4


L10  ANSWER 1 OF 4  USPATFULL
AN     2002:9647  USPATFULL
TI     Use of IL-12 and IL-12
       antagonists in the treatment of autoimmune diseases
IN     Leonard, John, Auburn, NH, United States
```

Goldman, Samuel, Acton, MA, United States
O'Hara, Jr., Richard, Quincy, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6338848 B1 20020115
AI US 2000-513380 20000225 (9)
RLI Continuation of Ser. No. US 1995-560943, filed on 20 Nov 1995, now
abandoned Continuation of Ser. No. US 1994-212629, filed on 14 Mar 1994,
now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Minnifield, Nita M.
LREP Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 676
AB Method of treating autoimmune conditions are disclosed comprising
administering to a mammalian subject **IL-12** or an **IL-12** antagonist. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels
of IFN-.gamma. or TNF-.alpha.. Suitable conditions for treatment include
multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and
autoimmune inflammatory eye disease.

L10 ANSWER 2 OF 4 USPATFULL
AN 2000:74115 USPATFULL
TI Polynucleotides encoding human CTLA-8 related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6074849 20000613
AI US 1996-685239 19960718 (8)
RLI Continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1658

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 related proteins are disclosed.
Human CTLA-8 proteins and methods for their production are also disclosed. Methods of treatment using human CTLA-8 proteins, rat CTLA-8
proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L10 ANSWER 3 OF 4 USPATFULL
AN 2000:37900 USPATFULL
TI Human CTLA-8 and uses of CTLA-8-related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6043344 20000328
AI US 1998-34810 19980304 (9)
RLI Division of Ser. No. US 1996-685239, filed on 18 Jul 1996, now abandoned
which is a continuation-in-part of Ser. No. US 1995-504032, filed on 19
Jul 1995 which is a continuation-in-part of Ser. No. US 1995-514014,
filed on 11 Aug 1995, now patented, Pat. No. US 5707829
PRAI US 1995-35347 19950719 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Lahive & Cockfield, LLP, Mandragouras, Esq., Amy E., Lauro, Esq., Peter
C.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1761
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 and related proteins are disclosed. Human CTLA-8 proteins and methods for their production are
also disclosed. Methods of treatment using human CTLA-8 proteins, rat
CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L10 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
AN 1995:934127 CAPLUS
DN 123:337469
TI Use of **IL-12** and **IL-12** antagonists in treatment of autoimmune diseases
IN **Leonard, John P.**; **Goldman, Samuel**; **O'Hara, Richard, Jr.**

PA Genetics Institute, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09510444	T2	19971021	JP 1995-524044	19950307
	US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		

AB Autoimmune conditions such as multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or TNF-.alpha., are treated in mammals by administering **IL-12** or an **IL-12** antagonist. Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with **IL-12** during restimulation, and alleviated by injection of a polyclonal antibody to **IL-12**.

=> d clm 1

L10 ANSWER 1 OF 4 USPATFULL
CLM What is claimed is:
1. A method for treating multiple sclerosis in a human subject, said method comprising administering to said subject a therapeutically effective amount of an **IL-12** antagonist that binds with **IL-12**, wherein said antagonist is selected from the group consisting of an antibody immunoreactive with **IL-**

12 and an antibody fragment immunoreactive with IL-12.

2. The method of claim 1, wherein said antagonist is administered in a dose of about 0.05 to about 25 mg/kg.

3. The method of claim 1, wherein said antagonist is administered in combination with a pharmaceutically acceptable carrier.

4. The method of claim 1, wherein said antagonist is an antibody immunoreactive with IL-12.

5. The method of claim 1, wherein said antagonist is an antibody fragment immunoreactive with IL-12.

```
=>  dhis

DHIS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> d his


     (FILE 'HOME' ENTERED AT 12:31:11 ON 18 JAN 2002)

     FILE 'EMBASE, MEDLINE, BIOSIS, USPATFULL, JAPIO, WPIDS, CAPLUS,
AGRICOLA,
     LIFESCI, BIOTECHDS, JICST-EPLUS' ENTERED AT 12:31:20 ON 18 JAN
2002
                  E LEONARD JOHN P/AU
L1             90 S E3-E5
                  E LEONARD J P/AU
L2            350 S E3-E4
                  E GOLDMAN SAMUEL/AU
L3             79 S E1-E9
                  E GOLDMAN S/AU
L4           1413 S E3
                  E OHARA RICHARD/AU
                  E O HARA RICHARD/AU
L5             25 S E3-E7
                  E O HARA R/AU
L6             78 S E3
L7             48 S E11
L8           2069 S L1-L7
L9             23 S L8 AND ARTHRITIS
L10             4 S L9 AND IL-12

=> dup rem l9

PROCESSING COMPLETED FOR L9
L11            12 DUP REM L9 (11 DUPLICATES REMOVED)
```

=> d bib ab 1-12

L11 ANSWER 1 OF 12 USPATFULL
AN 2002:9647 USPATFULL
TI Use of IL-12 and IL-12 antagonists in the treatment of autoimmune diseases
IN Leonard, John, Auburn, NH, United States
Goldman, Samuel, Acton, MA, United States
O'Hara, Jr., Richard, Quincy, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6338848 B1 20020115
AI US 2000-513380 20000225 (9)
RLI Continuation of Ser. No. US 1995-560943, filed on 20 Nov 1995, now abandoned Continuation of Ser. No. US 1994-212629, filed on 14 Mar 1994, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Minnifield, Nita M.
LREP Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 676
AB Method of treating autoimmune conditions are disclosed comprising administering to a mammalian subject IL-12 or an IL-12 antagonist. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels of IFN-.gamma. or TNF-.alpha.. Suitable conditions for treatment include multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and autoimmune inflammatory eye disease.

L11 ANSWER 2 OF 12 USPATFULL DUPLICATE 1
AN 2000:74115 USPATFULL
TI Polynucleotides encoding human CTLA-8 related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S.

corporation)
PI US 6074849 20000613
AI US 1996-685239 19960718 (8)
RLI Continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1658
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 related proteins are disclosed.

Human CTLA-8 proteins and methods for their production are also disclosed. Methods of treatment using human CTLA-8 proteins, rat CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L11 ANSWER 3 OF 12 USPATFULL DUPLICATE 2
AN 2000:37900 USPATFULL
TI Human CTLA-8 and uses of CTLA-8-related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6043344 20000328
AI US 1998-34810 19980304 (9)
RLI Division of Ser. No. US 1996-685239, filed on 18 Jul 1996, now abandoned which is a continuation-in-part of Ser. No. US 1995-504032, filed on 19 Jul 1995 which is a continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995, now patented, Pat. No. US 5707829
PRAI US 1995-35347 19950719 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Lahive & Cockfield, LLP, Mandragouras, Esq., Amy E., Lauro, Esq., Peter C.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1761
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 and related proteins are disclosed. Human CTLA-8 proteins and methods for their production are

also disclosed. Methods of treatment using human CTLA-8 proteins, rat
CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L11 ANSWER 4 OF 12 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD DUPLICATE
3
AN 2000-524532 [47] WPIDS
DNN N2000-387705 DNC C2000-155840
TI Humanized immunoglobulin having a binding specificity to B7-1 (derived
from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524) molecules,
modulates immune responses and can therefore treat e.g. autoimmune
diseases, infectious diseases.
DC B04 D16 S03
IN CARRENO, B; CELNIKER, A C; CO, M S; COLLINS, M; **GOLDMAN, S**;
GRAY, G S; KNIGHT, A; OHARA, D; RUP, B; VELDMAN, G M; O'HARA, D; VASQUEZ,
M
PA (GEMY) GENETICS INST INC
CYC 91
PI WO 2000047625 A2 20000817 (200047)* EN 158p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES
FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS
LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL
TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000039988 A 20000829 (200062)
NO 2001003911 A 20011010 (200174)
EP 1159300 A2 20011205 (200203) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
ADT WO 2000047625 A2 WO 2000-US3303 20000209; AU 2000039988 A AU 2000-39988
20000209; NO 2001003911 A WO 2000-US3303 20000209, NO 2001-3911 20010810;
EP 1159300 A2 EP 2000-919275 20000209, WO 2000-US3303 20000209
FDT AU 2000039988 A Based on WO 200047625; EP 1159300 A2 Based on WO 200047625
PRAI US 1999-339596 19990624; US 1999-249011 19990212
AB WO 200047625 A UPAB: 20000925
NOVELTY - Humanized immunoglobulin having a binding specificity to B7-1
(derived from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524)
molecules, comprising an antigen binding region of non-human origin and a
portion of a human immunoglobulin, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the

following:

(1) a host cell comprising nucleic acid that encodes a humanized B7-1 antibody and/or a humanized B7-2 antibody;

(2) a humanized immunoglobulin light/heavy chain having binding specificity for B7-1 comprising CDR1, CDR2, and CDR3 of the light/heavy chain of murine 1F1 antibody and a human light/heavy chain framework region;

(3) an isolated nucleic acid (N1) comprising a defined 390 base pair (bp) sequence, encoding a defined 130 amino acid human immunoglobulin light chain variable region (P1) of B7-1 (both given in the specification);

(4) an isolated nucleic acid (N2) comprising a defined 405 base pair (bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P2) of B7-1 (both given in the specification);

(5) an isolated nucleic acid (N3) comprising a defined 396 base pair (bp) sequence, encoding a defined 132 amino acid human immunoglobulin light chain variable region (P3) of B7-2 (both given in the specification);

(6) an isolated nucleic acid (N4) comprising a defined 405 base pair (bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P4) of B7-2 (both given in the specification);

(7) a fused gene encoding humanized immunoglobulin light or heavy chain comprising a first nucleic acid sequence encoding an antigen binding region derived from murine 1F1 or 3D1 monoclonal antibody and a second nucleic acid sequence encoding a portion of a constant region of an immunoglobulin of human origin;

(8) a method for inhibiting the interaction of a first cell bearing a B7-1 receptor with a second cell bearing B7-1, comprising contacting the first cell with a humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(9) a method for treating an individual having a transplanted organ, tissue or cell comprising administering humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(10) a method for treating a disease modulated by B7-1 or B7-2;

(11) a method for making a humanized immunoglobulin having binding

specificity for B7-1 or B7-2 comprising:

(a) determining the complementarity determining regions (CDRs) of an antibody of non-human origin which has binding specificity for B7-1 or B7-2;

(b) obtaining a human antibody having a framework region amino acid sequence suitable for grafting of the CDRs in (a); and

(c) grafting the CDRs of (a) with those of (b);

(12) a method for determining the presence or absence of B7-1 or B7-2 in a sample comprising:

(a) contacting the sample with an antibody specific to B7-1 or B7-2 to allow complex formation; and

(b) detecting the presence or absence of the complex;

(13) a humanized immunoglobulin light or heavy chain having binding specificity for B7-2 comprising CDR1, CDR2, and CDR3 of the light chain of murine 3D1 antibody, and a human light or heavy chain framework region;

(14) a method for transplanting cells into an individual comprising:

(a) obtaining cells from a donor;

(b) contacting the cells with an immunoglobulin specific to B7-1 and B7-2 and recipient cells from the individual to allow tolerance reduction; and

(c) introducing the mixture to the individual;

(15) a method for treating a disorder selected from autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, **arthritis**, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis comprising administering a humanized immunoglobulin to B7-1 and B7-2

(16) a method for treating a transplant recipient or preventing transplant rejection in a transplant recipient, comprising administering an immunoglobulin specific to B7-1 and B7-2; and

(17) a method for decreasing an antibody response to an antigen in a mammal comprising administering a humanized immunoglobulin specific to B7-1 or B7-2.

ACTIVITY - Immunosuppressive; antiinfective; antiinflammatory; dermatological; antidiabetic; antiasthmatic; antiarthritic; cytostatic; antianemic; neuroprotective.

MECHANISM OF ACTION - Modulation of immune responses; inhibition of T cell costimulation.

Isolated CD28+ T cells were washed once and resuspended in RPMI (not defined) complete medium, supplemented with 2 ng/ml PMA (not defined), to a cell density of 5 multiply 105 cells/ml. The CD28+ T cells were added to the antibody/CHO/hB7-2 mixture, incubated for 3 days at 37 deg. C, 5% CO2, and T cell proliferation was measured by pulsing for the last 6 hours of culture with 1 uCi of (3H)-thymidine. The cells were harvested on a filter and the incorporated radioactivity was measured in a scintillation counter. Results showed that both antibodies exhibited dose dependent inhibition of B7-2 driven T cell proliferation with similar IC50 (inhibitory concentration 50%) values of 72 pm (murine anti-hB7-2) and 50 pm (humanized anti-hB7-2) indicating that both antibodies were similar and very effective in inhibiting the B7-2 T cell stimulatory signal. This demonstrated that the high affinity anti-B7-2 mAbs could block B7-2 functionality by inhibiting the activation and/or proliferation of human T cells.

USE - The humanized immunoglobulin with binding specificity to B7-1 and/or B7-2 is useful for treating autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, arthritis, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis. The immunoglobulins are also useful for treating a transplant recipient or preventing transplant rejection in a transplant recipient, and treating proliferative disease (leukemia, lymphoma and cancer), anemia (sickle-cell anemia, thalassemia and aplastic anemia), inborn errors of metabolism, congenital immunodeficiency diseases, and myeloid dysplasia syndrome.

Dwg.0/28

L11 ANSWER 5 OF 12 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 2001107003 EMBASE

TI Acute-phase serum amyloid A production by rheumatoid **arthritis** synovial tissue.

AU **O'Hara R.**; Murphy E.P.; Whitehead A.S.; FitzGerald O.; Bresnihan B.

CS A.S. Whitehead, Univ. of Pennsylvania Sch. of Med., Philadelphia, PA, United States

SO Arthritis Research, (2000) 2/2 (142).
ISSN: 1465-9905 CODEN: ARRECG

CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
031 Arthritis and Rheumatism
LA English
SL English
AB Acute-phase serum amyloid A (A-SAA) is a major component of the acute-phase response. A sustained acute-phase response in rheumatoid **arthritis** (RA) is associated with increased joint damage. A-SAA mRNA expression was confirmed in all samples obtained from patients with RA, but not in normal synovium. A-SAA mRNA expression was also demonstrated in cultured RA synoviocytes. A-SAA protein was identified in the supernatants of primary synoviocyte cultures, and its expression colocalized with sites of macrophage accumulation and with some vascular endothelial cells. It is concluded that A-SAA is produced by inflamed RA synovial tissue. The known association between the acute-phase response and progressive joint damage may be the direct result of synovial A-SAA-induced effects on cartilage degradation.

L11 ANSWER 6 OF 12 MEDLINE
AN 2001154767 MEDLINE
DN 21062410 PubMed ID: 11062604
TI Acute-phase serum amyloid A production by rheumatoid **arthritis** synovial tissue.
AU **O'Hara R**; Murphy E P; Whitehead A S; FitzGerald O; Bresnihan B
CS St Vincent's University Hospital, Dublin, Ireland.
SO ARTHRITIS RESEARCH, (2000) 2 (2) 142-4.
Journal code: DWZ; 100913255. ISSN: 1465-9905.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010322
AB Acute-phase serum amyloid A (A-SAA) is a major component of the acute-phase response. A sustained acute-phase response in rheumatoid **arthritis** (RA) is associated with increased joint damage. A-SAA mRNA expression was confirmed in all samples obtained from patients with RA, but not in normal synovium. A-SAA mRNA expression was also demonstrated in cultured RA synoviocytes. A-SAA protein was identified in the supernatants of primary synoviocyte cultures, and its expression colocalized with sites of macrophage accumulation and with some vascular endothelial cells. It is concluded that A-SAA is produced by inflamed RA synovial tissue. The known association between the acute-phase response

and progressive joint damage may be the direct result of synovial A-SAA-induced effects on cartilage degradation.

L11 ANSWER 7 OF 12 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
DUPLICATE
4
AN 1997-132638 [12] WPIDS
CR 1997-165283 [15]; 2000-282570 [23]; 2000-430395 [36]
DNC C1997-042879
TI New nucleic acid encoding the CTLA-8 protein - modulates growth of
vascular endothelial and haematopoietic cells and induces cytokine
expression, for treating infection, auto-immune disease, etc.
DC B04 D16
IN CARLIN, M; JACOBS, K; KELLEHER, K; MCCOY, J M; GIANNOTTI, J; GOLDEN-FLEET,
M; **GOLDMAN, S**; MI, S; NEBEN, S; PITTMAN, D; DUCKETT, M C;
GOLDEN-FLEET, M M; PITMAN, D; CARLIN-DUCKETT, M
PA (GEMY) GENETICS INST INC
CYC 23
PI WO 9704097 A2 19970206 (199712)* EN 50p
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 9667123 A 19970218 (199723)
WO 9704097 A3 19970912 (199749)
US 5707829 A 19980113 (199809) 30p
EP 839196 A2 19980506 (199822) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 11510045 W 19990907 (199947) 59p
US 5969093 A 19991019 (199950)
MX 9800507 A1 19980501 (200007)
MX 9801120 A1 19990401 (200055)
AU 727480 B 20001214 (200103)
AU 727489 B 20001214 (200103)
AU 2001028001 A 20010517 (200138)#
AU 2001028002 A 20010802 (200152)#
ADT WO 9704097 A2 WO 1996-US11889 19960718; AU 9667123 A AU 1996-67123
19960218; US 5707829 A US 1995-514014 19950811; EP 839196 A2 EP
1996-927237 19960718, WO 1996-US11889 19960718; JP 11510045 W WO
1996-US11889 19960718, JP 1997-506846 19960718; US 5969093 A Div ex US
1995-514014 19950811, US 1997-833823 19970410; MX 9800507 A1 MX 1998-507
19980116; MX 9801120 A1 MX 1998-1120 19980210; AU 727480 B AU 1996-67123
19960718; AU 727489 B AU 1996-67685 19960808; AU 2001028001 A Div ex AU
1996-67685 19960808, AU 2001-28001 20010314; AU 2001028002 A Div ex AU
1996-67123 19960718, AU 2001-28002 20010314
FDT AU 9667123 A Based on WO 9704097; EP 839196 A2 Based on WO 9704097; JP
11510045 W Based on WO 9704097; AU 727480 B Previous Publ. AU 9667123,
Based on WO 9704097; AU 727489 B Previous Publ. AU 9667685, Based on WO
9707198; AU 2001028001 A Div ex AU 727489; AU 2001028002 A Div ex AU

727480
PRAI US 1995-514014 19950811; US 1995-504032 19950719; US 1997-833823
19970410; WO 1996-US12897 19960808; AU 2001-28001 20010314; AU
2001-28002 20010314
AB WO 9704097 A UPAB: 20011001
A novel isolated polynucleotide (I) comprises: (a) nucleotides (nt)
146-544 of an 813 bp sequence given in the specification; (b) a sequence
able to hybridise with (a) or varying from (a) only within the degeneracy
of the genetic code; or (c) an allelic variant of (a). Also claimed are:
(1) host cells transformed with (I); (2) isolated human CTLA-8 protein
which has 163 amino acids (aa), its 11-163, 29-163 or 31-163 regions or
any fragments of them with CTLA-8 activity; and (3) antibodies (Ab) which
specifically react with CTLA-8 protein.
USE - (I) encodes proteins with CTLA-8 activity. Treatment of mammals
with CTLA-8 (or non-human analogues or IL-17) results in at least one of:
(a) inhibition of angiogenesis, growth/proliferation of vascular
endothelial cells, tumour cells and angiogenesis-dependent tissue growth;
(b) proliferation of myeloid, erythroid or lymphoid cells (or their
progeny); or (c) induction of interferon- gamma , IL-3 or GM-CSF prodn
(claimed). Opt. CTLA-8 is expressed in vivo from a suitable vector.
Typical applications of CTLA-8 are treatment of immune deficiency and
disorders requiring modulation of T/B cell growth or proliferation, or of
cytolytic natural killer cells, e.g. viral or microbial infection (e.g.
HIV, hepatitis, malaria, candidiasis etc.); autoimmune disease (e.g.
multiple sclerosis, rheumatoid **arthritis**, insulin-dependent
diabetes etc.); to boost the immune response in cancer treatment; as
antiinflammatories (e.g. in septic shock or Crohn's disease) and in
haematopoietic disorders where growth/proliferation of erythroid, myeloid
or megakaryocytic cells is needed. Ab can be used to determine CTLA-8,
possibly also for treating some tumours or some of the above conditions.
Dwg.0/7

L11 ANSWER 8 OF 12 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD DUPLICATE
5

AN 1995-336810 [43] WPIDS
DNC C1995-148498
TI Use of interleukin-I2 or an Il-I2 antagonist - for treating autoimmune
conditions, eg. multiple sclerosis, lupus, rheumatoid **arthritis** or diabetes.
DC B04
IN **GOLDMAN, S**; **LEONARD, J P**; OHARA, R
PA (GEMY) GENETICS INST INC
CYC 23
PI WO 9524918 A1 19950921 (199543)* EN 37p
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
W: AU CA JP
AU 9519749 A 19951003 (199602)
ZA 9500960 A 19951227 (199605) 33p
EP 750509 A1 19970102 (199706) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
JP 09510444 W 19971021 (199801) 33p
AU 689236 B 19980326 (199826)
IL 112677 A 20000131 (200015)
TW 400233 A 20000801 (200109)
ADT WO 9524918 A1 WO 1995-US2550 19950307; AU 9519749 A AU 1995-19749 19950307; ZA 9500960 A ZA 1995-960 19950207; EP 750509 A1 EP 1995-912666
19950307, WO 1995-US2550 19950307; JP 09510444 W JP 1995-524044 19950307,
WO 1995-US2550 19950307; AU 689236 B AU 1995-19749 19950307; IL 112677 A
IL 1995-112677 19950216; TW 400233 A TW 1995-101380 19950214
FDT AU 9519749 A Based on WO 9524918; EP 750509 A1 Based on WO 9524918; JP
09510444 W Based on WO 9524918; AU 689236 B Previous Publ. AU 9519749,
Based on WO 9524918
PRAI US 1994-212629 19940314
AB WO 9524918 A UPAB: 19951102
A method for treating in a mammalian subject an autoimmune condition
comprises administering (i) an interleukin-I2 (IL-I2) antagonist or (ii)
IL-I2.
USE - The method is used partic. for autoimmune conditions which are
promoted by increased levels of TNF-alpha or IFN-gamma (claimed). It can
be used for treating multiple sclerosis, systemic lupus erythematosus,
rheumatoid **arthritis**, autoimmune pulmonary inflammation,
Ciuillan-Barre syndrome, autoimmune thyroiditis, insulin dependent
diabetes mellitus or autoimmune inflammatory eye disease (claimed).
Dwg.0/6

L11 ANSWER 9 OF 12 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 6
AN 92178943 EMBASE
DN 1992178943
TI .beta.-Adrenergic receptor density and function of peripheral blood

mononuclear cells are increased in multiple sclerosis: A regulatory role
for cortisol and interleukin-1.
AU Zoukos Y.; **Leonard J.P.**; Thomaides T.; Thompson A.J.; Cuzner M.L.
CS Multiple Sclerosis Society Lab., Institute of Neurology, 1, Wakefield
Street,London WC1N 1PJ, United Kingdom
SO Annals of Neurology, (1992) 31/6 (657-662).
ISSN: 0364-5134 CODEN: ANNED3
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
025 Hematology
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB An increased density of .beta.-adrenergic receptors was demonstrated on
peripheral blood mononuclear cells (PBMCs) from patients with progressive
or relapsing-remitting multiple sclerosis (MS). The same observation was
made in patients with chronic active rheumatoid **arthritis**, but
not in those with myasthenia gravis. The affinity of the receptors was
within the normal range in all tested groups of patients and there was a
positive correlation between density and function as determined by
intracellular cyclic AMP production after stimulation with isoproterenol.
A putative link between inflammatory processes and the functional
upregulation of .beta.-adrenergic receptors on PBMCs was tested by in
vitro studies with the soluble mediators interleukin-1 and hydrocortisone.
A functional upregulation of .beta.-adrenergic receptors was observed when
PBMCs from normal control subjects were cultured in the presence of either
mediator, whereas the already upregulated receptor density on PBMCs from
patients with MS remained unchanged. Whether this represents a recovery
mechanism to inflammation in MS or a blunting of homeostatic
immunoregulatory mechanisms requires further investigation.

L11 ANSWER 10 OF 12 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92231358 EMBASE
DN 1992231358
TI Observations, legends, and conjectures concerning restricted T-cell
receptor usage and autoimmune disease.
AU Esch T.; Clark L.; Zhang X.-M.; **Goldman S.**; Heber-Katz E.
CS Wistar Institute, 3601 Spruce Street,Philadelphia, PA 19104, United States
SO Critical Reviews in Immunology, (1991) 11/5 (249-264).

ISSN: 1040-8401 CODEN: CCRIDE
CY United States
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB It has become clear over the past few years that a variety of experimental autoimmune conditions are mediated by T cells bearing a highly restricted subset of antigen receptors. This restricted TcR usage raises important questions concerning not only the recognition of autoantigens, but also the pathogenic mechanisms underlying many models of autoimmunity. Furthermore, the extension of these findings in certain cases to human disease has raised the possibility of specific therapeutic immune intervention. In this review, we examine the available data on restricted T-cell receptor usage in autoimmune disorders and explore the interpretations and the theoretical and practical implications of these findings.

L11 ANSWER 11 OF 12 MEDLINE DUPLICATE 7
AN 78126423 MEDLINE
DN 78126423 PubMed ID: 630979
TI Rheumatoid pericarditis presenting as a mass lesion.
AU **Goldman S**; Gall E P; Hager W D
SO CHEST, (1978 Apr) 73 (4) 550-2.
Journal code: D1C; 0231335. ISSN: 0012-3692.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197805
ED Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19780524

L11 ANSWER 12 OF 12 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 78368952 EMBASE
DN 1978368952
TI Rheumatoid pericarditis presenting as a mass lesion.
AU **Goldman S.**; Gall E.P.; Hager W.D.
CS Cardiol. Sect., Dept. Med., VA Hosp., Tucson, Ariz., United States
SO Chest, (1978) 73/4 (550).
CODEN: CHETBF
CY United States
DT Journal
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
031 Arthritis and Rheumatism
014 Radiology
LA English

AB The findings of a loculated pericardial effusion presenting as a mass
lesion are described in a 54-year-old man with rheumatoid **arthritis** who exhibited findings of both cardiac tamponade and of constrictive pericarditis.

=> s l8 and (il-12 or nksf or clmf)

L12 60 L8 AND (IL-12 OR NKSF OR CLMF)

=> dup rem l12

PROCESSING COMPLETED FOR L12
L13 24 DUP REM L12 (36 DUPLICATES REMOVED)

=> d bib ab 1-24

L13 ANSWER 1 OF 24 USPATFULL
AN 2002:9647 USPATFULL
TI Use of **IL-12** and **IL-12** antagonists in the treatment of autoimmune diseases
IN Leonard, John, Auburn, NH, United States
Goldman, Samuel, Acton, MA, United States
O'Hara, Jr., Richard, Quincy, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6338848 B1 20020115
AI US 2000-513380 20000225 (9)
RLI Continuation of Ser. No. US 1995-560943, filed on 20 Nov 1995, now abandoned Continuation of Ser. No. US 1994-212629, filed on 14 Mar 1994, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Minnifield, Nita M.
LREP Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 676
AB Method of treating autoimmune conditions are disclosed comprising administering to a mammalian subject **IL-12** or an **IL-12** antagonist. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels of IFN-.gamma. or TNF-.alpha.. Suitable conditions for treatment include multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and autoimmune inflammatory eye disease.

L13 ANSWER 2 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 1

AN 2001214972 EMBASE
TI Myelin/axonal pathology in interleukin-12 induced serial relapses of experimental allergic encephalomyelitis in the Lewis rat.
AU Ahmed Z.; Gveric D.; Pryce G.; Baker D.; **Leonard J.P.**; Cuzner M.L.
CS Dr. Z. Ahmed, Miriam Marks Dept. of Neurochemistry, Institute of Neurology, University College London, 1 Wakefield Street, London, WC1N 1PJ, United Kingdom. z.ahmed@ion.ucl.ac.uk
SO American Journal of Pathology, (2001) 158/6 (2127-2138).
Refs: 57
ISSN: 0002-9440 CODEN: AJPAA4
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
AB Lewis rats, on recovery from monophasic clinical experimental allergic encephalomyelitis (EAE), can be induced to develop repeated paralytic relapses with a graded reduction in clinical severity following intraperitoneal administration of **IL-12**. By the time of the third relapse, the number and size of inflammatory cuffs in the spinal cord were reduced with the makeup of the cellular infiltrate shifting to a significantly increased number of B cells. Serum levels of myelin basic protein (MBP)-specific IgG1 and IgG2b were found to rise over time while MBP and MBP peptide-positive macrophages and microglia became evident in perivascular cuffs and in spinal cord parenchyma, indicative of myelin phagocytosis. Axonal death was observed in semithin and EM sections of spinal cord in third relapse animals in association with iNOS and tPA immunostaining throughout gray and white matter. These neurotoxic or excitotoxic agents may contribute to axonal damage directly or indirectly by activated microglia and macrophages, leading to limited damage of the axonal-myelin unit.

L13 ANSWER 3 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 2
AN 2001274332 EMBASE
TI Interleukin-12 gene therapy vaccines: Directing the immune system against minimal residual leukemia.
AU Dunussi-Joannopoulos K.; **Leonard J.P.**
CS Dr. K. Dunussi-Joannopoulos, Genetics Institute, One Burtt Road, Andover, MA 01810, United States

SO Leukemia and Lymphoma, (2001) 41/5-6 (483-492).
Refs: 60
ISSN: 1042-8194 CODEN: LELYEA
CY United Kingdom
DT Journal; General Review
FS 016 Cancer
022 Human Genetics
025 Hematology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Current overall survival rates for patients with AML remain poor and there is need for novel therapeutic approaches. One such approach is to use the patient's own immune system to eliminate minimal residual disease. Recent advances in genetic manipulation of tumor cells, together with a better understanding of the immune mechanisms controlling the host-tumor relationship have led to a flurry of preclinical and clinical studies on tumor cell vaccines. Here we present a brief overview of genetic manipulation of tumor cells, and highlight important principles of cancer immunity and cancer vaccines. Special emphasis is given on recent work on the role of interleukin-12 (**IL-12**) based vaccines in murine AML. These studies have shown that vaccines with AML cells, genetically modified to secrete **IL-12**, are potent stimulators of the immune system and lead to the development of both prophylactic and therapeutic anti-leukemia immunity.

L13 ANSWER 4 OF 24 USPATFULL
AN 2000:74115 USPATFULL
TI Polynucleotides encoding human CTLA-8 related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6074849 20000613
AI US 1996-685239 19960718 (8)
RLI Continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.

CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1658
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 related proteins are disclosed.
Human CTLA-8 proteins and methods for their production are also disclosed. Methods of treatment using human CTLA-8 proteins, rat CTLA-8
proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L13 ANSWER 5 OF 24 USPATFULL
AN 2000:37900 USPATFULL
TI Human CTLA-8 and uses of CTLA-8-related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6043344 20000328
AI US 1998-34810 19980304 (9)
RLI Division of Ser. No. US 1996-685239, filed on 18 Jul 1996, now abandoned
which is a continuation-in-part of Ser. No. US 1995-504032, filed on 19
Jul 1995 which is a continuation-in-part of Ser. No. US 1995-514014,
filed on 11 Aug 1995, now patented, Pat. No. US 5707829
PRAI US 1995-35347 19950719 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Lahive & Cockfield, LLP, Mandragouras, Esq., Amy E., Lauro, Esq., Peter
C.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1761
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 and related proteins are disclosed. Human CTLA-8 proteins and methods for their production are
also disclosed. Methods of treatment using human CTLA-8 proteins, rat
CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L13 ANSWER 6 OF 24 BIOSIS COPYRIGHT 2002 BIOSIS
AN 2001:39565 BIOSIS
DN PREV200100039565

TI Myelin oligodendrocyte glycoprotein induced EAE in **IL-12** p35 deficient mice.
AU Hunter, S. E. (1); Thibodeaux, D. K. (1); Bouchard, P. (1); **Leonard, J. P. (1)**
CS (1) Genetics Institute, Inc., Cambridge, MA, 02140 USA
SO FASEB Journal, (April 20, 2000) Vol. 14, No. 6, pp. A1116. print.
Meeting Info.: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA May 12-16, 2000
ISSN: 0892-6638.
DT Conference
LA English
SL English

L13 ANSWER 7 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 3
AN 1999402567 EMBASE
TI Autocrine regulation of **IL-12** receptor expression is independent of secondary IFN-.gamma. secretion and not restricted to T and NK cells.
AU Thibodeaux D.K.; Hunter S.E.; Waldburger K.E.; Bliss J.L.; Trepicchio W.L.; Sypek J.P.; Dunussi-Joannopoulos K.; Goldman S.J.; **Leonard J.P.**
CS Dr. J.P. Leonard, Genetics Institute, Preclinical RandD, One Burtt Road, Andover, MA 01810, United States. jleonard@genetics.com
SO Journal of Immunology, (15 Nov 1999) 163/10 (5257-5264).
Refs: 39
ISSN: 0022-1767 CODEN: JOIMA3
CY United States
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
AB The biological response to **IL-12** is mediated through specific binding to a high affinity receptor complex composed of at least two subunits (designated IL-12R.beta.1 and IL-12R.beta.2) that are expressed on NK cells and activated T cells. The selective loss of IL-12R.beta.2 expression during Th2 T cell differentiation suggests that regulation of this receptor component may govern **IL-12** responsiveness. In murine assays, down-regulation of IL-12R.beta.2 expression can be prevented by treatment with IFN-.gamma., indicating that receptor expression and hence **IL-12** responsiveness may be regulated, at least in part, by the local cytokine milieu. In this study, we report that cellular expression of both IL-12R.beta.1 and

.beta.2 mRNA is increased in the lymph nodes of naive mice following systemic administration of murine rIL-12 (rmIL-12). Changes in IL-12R mRNA were associated with increased IFN-.gamma. secretion following ex vivo activation of lymph node cells with rmIL-12, indicating the presence of a functional receptor complex. Expression of IL-12R mRNA was not restricted to lymph node T cells, and its autocrine regulation was independent of secondary IFN-.gamma. secretion. Data from fractionated lymph node cells as well as rmIL-12-treated B cell-deficient mice suggest that **IL-12**- responsive B cells may represent an alternative cellular source for IFN-.gamma. production. However, the strength of the biological response to rmIL-12 is not governed solely by receptor expression, as rmIL-12-induced IFN-.gamma. secretion from cultured lymph node cells is accessory cell dependent and can be partially blocked by inhibition of B7 costimulation.

L13 ANSWER 8 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 4
AN 1999432005 EMBASE
TI Vaccines with interleukin-12-transduced acute myeloid leukemia cells elicit very potent therapeutic and long-lasting protective immunity.
AU Dunussi-Joannopoulos K.; Runyon K.; Erickson J.; Schaub R.G.; Hawley R.G.; **Leonard J.P.**
CS Dr. K. Dunussi-Joannopoulos, Genetics Institute, 1 Burtt Rd, Andover, MA 01810, United States
SO Blood, (1999) 94/12 (4263-4273).
Refs: 66
ISSN: 0006-4971 CODEN: BLOOAW
CY United States
DT Journal; Article
FS 025 Hematology
037 Drug Literature Index
LA English
SL English
AB Interleukin-12 (**IL-12**) is a heterodimeric cytokine mediating a dynamic interplay between T cells and antigen-presenting cells (APCs). Preclinical studies have demonstrated that recombinant murine **IL-12** (rmIL-12) promotes specific antitumor immunity mediated by T cells in several types of tumors. However, the in vivo antitumor properties of **IL-12** in acute myeloid leukemia (AML) have not been previously reported. We show here in a murine

AML model that systemic administration of rmIL-12 significantly delays tumor growth but is incapable of rescuing mice from lethal leukemia. In contrast, AML cells genetically modified to express **IL-12** (IL12-AML) using murine stem cell virus (MSCV) p40 + p35 elicit very potent antileukemic activity. Vaccines with lethally irradiated IL12-AML cells protect naive mice against challenge with wild-type AML cells and, more importantly, can cure mice bearing a considerable leukemic burden. Immunized mice show no signs of systemic **IL-12** toxicity and their spleen histology is comparable with naive mice spleen. In vivo depletion of **IL-12**, interferon-.gamma. (IFN-.gamma.), or CD8+ T cells after injections with live IL12-AML cells abrogates completely the antileukemia immune responses. Studies on the in vitro effects of IFN-.gamma. on AML cells demonstrate enhanced expression of major histocompatibility complex (MHC) and accessory molecules and induction of the costimulatory molecules B7.1 and B7.2, but no significant direct antiproliferative effect. 51Cr release assays show that rejection of live IL12-AML cells supports the development of long-lasting leukemia-specific cytotoxic T lymphocyte (CTL) activity. In conclusion, our results demonstrate that IL12-AML vaccination is a safe and potent immunotherapeutic approach that has a great potential to eliminate minimal residual disease in patients with AML.

L13 ANSWER 9 OF 24 BIOSIS COPYRIGHT 2002 BIOSIS
AN 1999:275655 BIOSIS
DN PREV199900275655
TI Prolonged inhibition of murine lupus by short term therapy with anti-B7 and anti-**IL-12** antibodies during onset of disease.
AU Collins, M. (1); Nagle, S. (1); Chung, C. (1); **Goldman, S. (1)**; Sypek, J. (1)
CS (1) Genetics Institute, Andover, MA, 01810 USA
SO FASEB Journal, (March 15, 1999) Vol. 13, No. 5 PART 2, pp. A956. Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 99 Washington, D.C., USA April 17-21, 1999 Federation of American Societies for Experimental Biology . ISSN: 0892-6638.
DT Conference
LA English

L13 ANSWER 10 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 5

AN 1999315480 EMBASE
TI Immunological reconstitution and correlation of circulating serum inflammatory mediators/cytokines with the incidence of acute graft-versus-host disease during the first 100 days following unrelated umbilical cord blood transplantation.
AU Abu-Ghosh A.; **Goldman S.**; Slone V.; Van de Ven C.; Suen Y.; Murphy L.; Sender L.; Cairo M.S.
CS Dr. M.S. Cairo, Georgetown University Medical Center, Lombardi Cancer Center, 2 East Main, 3800 Reservoir Rd. NW, Washingon DC 20007, United States
SO Bone Marrow Transplantation, (1999) 24/5 (535-544).
Refs: 39
ISSN: 0268-3369 CODEN: BMTRE
CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
AB We investigated early immunological reconstitution and the production of circulating inflammatory mediators and their relationship to aGVHD in children during the first 100 days following unrelated UCBT. Nine patients had an underlying malignant disease (ALL, ANLL), and two, non-malignant diseases (SAA, ALD). The median age was 10 years (range: 1.25-21). Seven of 11 patients were alive by day 100, two died from regimen-related toxicity, and two died from severe aGVHD (grade .gtoreq. III). Myeloid engraftment (ANC .gtoreq. 500 /mm3 x 2 days) occurred at a median of 24 days (range: 14-55), while platelet engraftment (platelet count .gtoreq. 20,000 /mm3 untransfused x 7 days) was delayed and occurred at a median of 52 days (range: 33-95). The mean cell dose of CD34+ cells was 3.3 .+-. 3.51 x 105 /kg, and of CD34+/CD41+ cells was 3.94 .+-. 3.99 x 104 /kg. Acute GVHD (grade II-IV) developed in seven patients (77%), and severe aGVHD (grade III-IV) developed in five patients (55%). Serum levels of IL-2R.alpha.; IL-2, IL-4, IL-7, **IL-12**, and IFN.gamma. were not significantly different between patients with grades 0-I aGVHD and patients with grades II-IV aGVHD. Evaluation of immunological reconstitution on day 90 post UCBT demonstrated an early recovery of the

absolute numbers of B cells (CD19+) and NK cells (CD3-/CD56+). Immunoglobulin levels for IgG, IgM and IgA remained normal throughout the study period. PMN functional studies demonstrated normal superoxide generation, bacterial killing (BK), and chemotaxis (CTX). However, both helper (CD3+/CD4+) and suppressor (CD3+/CD8+)T cell subsets remained low during the first 100 days post UCBT with mean .+-. s.e.m. values of 120 .+-. 29 /mm3 and 10 .+-. 50 /mm3, respectively (normal = 900-2860 /mm3 (CD3/CD4), normal = 630-1910 /mm3 (CD3/CD8)). Mitogen response studies showed low blastogenesis to PHA and PWM, with a mean c.p.m. .+-. s.e.m. value of 1.7 .+-. 0.67 x 104 for PHA (NL .gtoreq. 75 x 103) and 8.42 .+-. 4.1 x 103 for PWM (NL .gtoreq. 25 x 103). In conclusion, serum levels of inflammatory mediators were not predictive nor did they correlate with the severity of aGVHD. Recovery of NK cells, B cells, and PMN functions occurred within the first 90 days post transplant. However, T cell subsets, CD3+/CD4+ and CD3+/CD8+, and T cell functional activity remained significantly decreased and may account for the high incidence of infectious morbidity seen during this immediate post UCBT period.

L13 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2002 ACS
AN 2000:177320 CAPLUS
DN 133:191823
TI Dose and timing of interleukin (**IL**)-**12** and timing and type of total-body irradiation: effects on graft-vs.-host disease inhibition and toxicity of exogenous **IL-12** in murine bone marrow transplant recipients
AU Sykes, Megan; Pearson, Denise A.; Taylor, Patricia A.; Szot, Gregory L.; **Goldman, Samuel J.**; Blazar, Bruce R.
CS BMT Section, Transplantation Biology Research Center, Surgical Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, 02129, USA
SO Biol. Blood Marrow Transplant. (1999), 5(5), 277-284
CODEN: BBMTF6; ISSN: 1083-8791
PB Carden Jennings Publishing
DT Journal
LA English
AB Paradoxically, a single injection of recombinant murine interleukin (**IL**)-**12** on the day of bone marrow transplantation (BMT) inhibits graft-vs.-host disease (GVHD) while preserving graft-vs.-leukemia (GVL) effects in lethally irradiated mice receiving fully MHC-mismatched

bone marrow and spleen cells. These protective effects are mediated by interferon (IFN)-.gamma., whose early secretion is induced by **IL-12** treatment. We investigated the relationship of **IL-12** dose and timing of administration, as well as timing and type of total-body irradn. (TBI), with the ability of **IL-12** to inhibit GVHD or mediate toxicity. A relatively low dose of **IL-12** (as little as 50 U in a single injection) can mediate significant GVHD protection. The timing of **IL-12** administration, however, is a crit. factor. **IL-12** administered 1 h before BMT was most protective, but protection was still obsd. when it was administered 1-12 h after BMT. Delaying **IL-12** administration to 36 h post-BMT completely obviated its protective effect. Administration of a second **IL-12** injection 6 days after BMT negated the protective effect of an initial injection at the time of BMT. While **IL-12** protection was evident when TBI was administered by 137Cs-irradiator in one or two fractions on day -1 or day 0, the use of an X-irradiator to deliver TBI on day -1 was assocd. with marked **IL-12** toxicity. Whereas the protective effect of **IL-12** against GVHD depended on donor-derived IFN-.gamma., toxicity depended on the ability of host cells to produce IFN-.gamma.. Careful studies are warranted to test the effects of **IL-12** in the context of BMT with various conditioning regimens in large animal preclin. models before this novel approach to GVHD protection can be applied clin.

RE.CNT 33

RE

(1) Allen, R; Eur J Immunol 1993, V23, P333 CAPLUS
(2) Atkins, M; Clin Cancer Res 1997, V3, P409 CAPLUS
(3) Berger, M; Transplantation 1994, V57, P1095 CAPLUS
(4) Blazar, B; J Immunol 1997, V158, P29 CAPLUS
(5) Blazar, B; Transplantation 1997, V64, P571 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 12 OF 24 CAPLUS COPYRIGHT 2002 ACS

AN 1998:565295 CAPLUS
DN 129:314782

TI Advanced colorectal cancer is associated with impaired interleukin 12 and enhanced interleukin 10 production

AU **O'Hara, Richard J.**; Greenman, John; MacDonald, Alistair W.; Gaskell, Kay M.; Topping, Katherine P.; Duthie, Graeme S.; Kerin, Michael J.; Lee, Peter W. R.; Monson, John R. T.
CS Academic Surgical Unit, The University of Hull, East Yorkshire, HU16 5JQ, UK

SO Clin. Cancer Res. (1998), 4(8), 1943-1948
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal

LA English
AB Interleukin 12 (**IL-12**) is a heterodimeric cytokine that has been demonstrated to have a major role in stimulating a cell-mediated antitumor response. IL-10, a product of T helper 2 lymphocytes, is its most potent inhibitor. The aim of this study was to investigate whether patients with colorectal cancer had an imbalance in prodn. of **IL-12** and IL-10 preoperatively, and whether this was assocd. with advanced disease at surgery. Blood was obtained before surgery from 60 patients with colorectal cancer and from 30 controls. Peripheral blood mononuclear cells were incubated with Staphylococcus aureus Cowan's strain 1 in vitro for 24 h to assess **IL-12** expression after stimulation, and serum was used for IL-10 measurement. **IL-12** and IL-10 levels were assessed by ELISA. A single pathologist staged the tumors according to the tumor-node-metastasis (TNM) and Dukes' classifications. Patients with colorectal cancer had significantly lower levels of **IL-12** ($P < 0.001$) and higher levels of IL-10 ($P = 0.004$) compared to controls. In addn., lower levels of **IL-12** were detected in those patients who were node pos. ($P < 0.05$), had Dukes' C lesions (P .ltoreq. 0.001), and T3 or T4 lesions ($P < 0.033$) when compared to controls. Patients with Dukes' B and C lesions ($P < 0.01$) and T3 and T4 lesions ($P < 0.05$) also had higher levels of IL-10 compared to controls. This study is the first to demonstrate that patients with colorectal cancer have decreased **IL-12** prodn. and increased serum IL-10. This suggests an impaired T helper 1 cell-mediated antitumor response and provides some justification for exogenous **IL-12** therapy or anti-IL-10 therapy in these patients.

L13 ANSWER 13 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 6
AN 1998011431 EMBASE
TI Immunoregulation by interleukin-12 in MB49.1 tumor-bearing mice: Cellular and cytokinemediated effector mechanisms.
AU Hunter S.E.; Waldburger K.E.; Thibodeaux D.K.; Schaub R.G.; Goldman S.J.; **Leonard J.P.**
CS J.P. Leonard, Genetics Institute, One Burtt Road, Andover, MA 01810, United States
SO European Journal of Immunology, (1997) 27/12 (3438-3446).
Refs: 33
ISSN: 0014-2980 CODEN: EJIMAF
CY Germany
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
LA English

SL English
AB Administration of recombinant murine interleukin (rmIL)-12 to MB49.1
tumor-bearing mice results in dose-dependent regression of the primary
tumor and the generation of protective antitumor immunity in the majority
of animals. rmIL-12 administration is associated with a marked increase in
lymph node cellularity that is predominantly due to the expansion of B220+
B cells as well as CD8+ T cells. Stimulation of lymph node cells from
rmIL-12-treated, but not control tumor-bearing mice, with MB49.1 tumor
cells in vitro was shown to enhance the secretion of interferon
(IFN)-.gamma.. The magnitude of this in vitro response was dependent on
the dose of rmIL-12 administered in vivo and mirrored the change in
circulating serum IFN-.gamma.. Furthermore, at the height of the in vitro
response to tumor stimulation, the addition of a neutralizing antibody to
murine **IL-12** suppressed IFN-.gamma. production,
indicating a role for endogenous **IL-12** in this
antigen-specific cytokine response. Although studies in SCID mice
confirmed that an appropriate T cell response was required for
rmIL-12-mediated antitumor activity, in immunocompetent animals early
tumor regression was not accompanied by cellular infiltration of the
tumor. In contrast, a profound increase in tumor-associated inducible
nitric oxide synthase (iNOS) was observed in mice receiving rmIL-12 which
preceded T cell infiltration of the tumor which could be detected during
the second week of **IL-12** treatment. Direct tumor
killing through the cytotoxic actions of NO via the iNOS pathway may serve
as a way of generating tumor antigen which enables the host to mount a
subsequent T cell response against the tumor.

L13 ANSWER 14 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 7
AN 97297122 EMBASE
DN 1997297122
TI Effects of single-dose interleukin-12 exposure on interleukin-12
associated toxicity and interferon-.gamma. production.
AU **Leonard J.P.**; Sherman M.L.; Fisher G.L.; Buchanan L.J.; Larsen
G.; Atkins M.B.; Sosman J.A.; Dutcher J.P.; Vogelzang N.J.; Ryan J.L.
CS Dr. J.L. Ryan, Genetics Institute, 87 Cambridge Park Dr, Cambridge, MA
02140, United States
SO Blood, (1997) 90/7 (2541-2548).
Refs: 33

ISSN: 0006-4971 CODEN: BLOOAW
CY United States
DT Journal; Article
FS 016 Cancer
025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Interleukin-12 (**IL-12**) is a key regulator of cell-mediated immunity that has therapeutic potential in cancer and infectious disease. In a previous Phase I dose escalation study of a single test dose of recombinant human **IL- 12** (rhIL-12) followed 14 days later by cycles of five consecutive daily intravenous injections every 3 weeks, we showed that a dose level up to 500 ng/kg could be administered with acceptable levels of safety. Based on these results, a Phase 2 study was conducted. In the Phase 2 study, however, administration of rhIL-12 at this same dose level resulted in severe toxicities with some patients unable to tolerate more than two successive doses. Of the 17 patients receiving rhIL-12 in the Phase 2 study, 12 patients were hospitalized and two patients died. A thorough scientific investigation to determine the cause of this unexpected toxicity failed to identify any difference in the drug products used or the patient populations enrolled in the Phase 1 and Phase 2 studies that could have accounted for the profound difference in toxicity. The focus of the investigation therefore shifted to the schedule of rhIL-12 administration.

We determined that a single injection of rhIL-12 2 weeks before consecutive dosing included in the Phase 1 study, but not in the schedule of administration in the Phase 2 study, has a profound abrogating effect on **IL-12**-induced interferon-.gamma. (IFN-.gamma.) production and toxicity. This observation of schedule-dependent toxicity of **IL-12** has been verified in mice, as well as nonhuman primates. In this regard, a single injection of **IL-12** before consecutive daily dosing protected mice and cynomolgus monkeys from acute toxicity including mortality and was associated with an attenuated IFN-.gamma. response. Because of this unique biologic response, careful attention to the schedule of administration is required to assure safe and effective clinical development of this highly promising cytokine.

L13 ANSWER 15 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 8
AN 97166329 EMBASE
DN 1997166329
TI Interleukin-12 induces relapse in experimental allergic encephalomyelitis in the Lewis rat.
AU Smith T.; Hewson A.K.; Kingsley C.I.; **Leonard J.P.**; Cuzner M.L.
CS Dr. T. Smith, Multiple Sclerosis Laboratory, Miriam Marks Dept. of Neurochemistry, Institute of Neurology, 1 Wakefield Street, London WC1N 1PJ, United Kingdom
SO American Journal of Pathology, (1997) 150/6 (1909-1917).
Refs: 51
ISSN: 0002-9440 CODEN: AJPAA4
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
AB Acute, monophasic experimental allergic encephalomyelitis (EAE) in the Lewis rat shows pathological similarities to the human disease multiple sclerosis (MS). Rats that recover from EAE are essentially resistant to disease reinduction, unlike MS in which relapses are frequently associated with common bacterial and viral infections. As macrophage-derived interleukin (**IL**)-**12** is a critical component of innate resistance to bacterial infection and appears to directly activate encephalitogenic T cells in vivo, the ability of this cytokine to reinduce paralysis in EAE was examined. Paralytic disease was exacerbated by intraperitoneal **IL-12** administration and could be reinduced up to 1 week after recovery from the primary clinical episode. Concomitant with worsening of initial clinical signs and relapse was an increase in the ratio of macrophages to T cells in brain stem perivascular cuffs and the expression of inducible nitric oxide synthase in cells with both macrophage anti microglial morphology. These findings suggest that **IL- 12** may contribute to macrophage-mediated disease exacerbation and relapse in patients with MS.

L13 ANSWER 16 OF 24 BIOSIS COPYRIGHT 2002 BIOSIS
AN 1998:69602 BIOSIS
DN PREV199800069602
TI Regulation of **IL-12** receptor expression and ex vivo cytokine production following rmIL-12 administration to C57BL/6 mice.

AU Thibodeaux, D.; Hunter, S. E.; Trepicchio, W. L.; Kobayashi, M.; **Leonard, J. P.**
CS Genet. Inst., Andover, MA 01810 USA
SO Cytokine, (Nov., 1997) Vol. 9, No. 11, pp. 963.
Meeting Info.: Fifth Annual Conference of the International Cytokine
Society Lake Tahoe, Nevada, USA November 9-13, 1997 International Cytokine
Society
. ISSN: 1043-4666.
DT Conference
LA English

L13 ANSWER 17 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 9
AN 97205418 EMBASE
DN 1997205418
TI Suppression of cyclophosphamide induced diabetes development and pancreatic Th1 reactivity in NOD mice treated with the interleukin (**IL**)-**12** antagonist **IL-12**[p40)2.
AU Rothe H.; **O'Hara R.M. Jr.**; Martin S.; Kolb H.
CS Dr. H. Rothe, Diabetes Research Institute, Auf'm Hennekamp 65, D-40225
Dusseldorf, Germany
SO Diabetologia, (1997) 40/6 (641-646).
Refs: 38
ISSN: 0012-186X CODEN: DBTGAJ
CY Germany
DT Journal; Article
FS 003 Endocrinology
026 Immunology, Serology and Transplantation
LA English
SL English
AB The macrophage product interleukin (**IL**)-**12** is known to drive Th1 reactions in physiological and pathological immune responses.
Here we report that treatment with the homodimeric IL-12p40 subunit, an antagonist of the bioactive IL-12p35/p40 heterodimer, suppresses diabetes development in cyclophosphamide-injected NOD mice. Female mice of 70 days old received cyclophosphamide (250 mg/kg) to accelerate and synchronize diabetes development, and daily injections of 1 .mu.g **IL-12**(p40)2. While there was no delay of the first diabetes cases, the incidence of overt diabetes was significantly decreased in treated mice (46 vs 23%, $p < 0.05$). Analysis of mRNA expression in the pancreas showed that administration of the **IL-12** antagonist had dampened interferon-gamma gene expression, decreased the ratio of interferon-gamma/IL-10 mRNA levels and in parallel suppressed the expression of the inducible nitric oxide synthase. At the same time intra-islet infiltration was significantly decreased ($p < 0.001$). Interestingly, the administration of **IL-12**(p40)2 also affected

IL-12 gene expression, by downregulation of p35 mRNA. We conclude that **IL-12** p40 homodimer suppresses diabetes development in the NOD mouse by dampening islet inflammation via selective down-regulation of Th1 type responses. The naturally occurring **IL - 12** antagonist **IL-12**(p40)2 represents a new and specific Th1 directed approach to prevent autoimmune diabetes.

L13 ANSWER 18 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 10
AN 97383463 EMBASE
DN 1997383463
TI Regulation of the inflammatory response in animal models of multiple sclerosis by interleukin-12.
AU **Leonard J.P.**; Waldburger K.E.; Schaub R.G.; Smith T.; Hewson A.K.; Cuzner M.L.; Goldman S.J.
CS J.P. Leonard, Genetics Institute, Preclinical Pharmacology, Andover, MA 01810, United States
SO Critical Reviews in Immunology, (1997) 17/5-6 (545-553).
Refs: 54
ISSN: 1040-8401 CODEN: CCRIDE
CY United States
DT Journal; Conference Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
LA English
SL English
AB Interleukin 12 (**IL-12**), a novel heterodimeric protein produced primarily by antigen-presenting cells, serves as a key regulator of innate and adaptive immune responses. In addition to being a potent inducer of IFN-.gamma., **IL-12** is widely considered to be the principal cytokine that regulates the generation of Th1 type effector cells. As the successful induction of experimental autoimmune encephalomyelitis (EAE) is associated with a strong Th1 type cellular response, we have evaluated the role of **IL-12** in regulating the pathogenesis of EAE in SJL/J mice and Lewis rats. In both settings, treatment with **IL-12** was found to accelerate the onset and increase the severity and duration of clinical disease. More importantly, administration of **IL-12** to Lewis rats that had recovered from primary disease was found to trigger clinical relapse. In all instances, **IL-12**-induced exacerbation was associated with a profound increase in iNOS positive macrophages within the perivascular lesions. Although **IL-12** -induced IFN-.gamma. does not appear to be required for exacerbation of disease, neutralizing antibodies against murine **IL-12**

delay the onset and reduce the severity of adoptively transferred EAE,
indicating a role for endogenous **IL-12** as regulator of disease. Based on the above findings, effective inhibition of **IL-12** in vivo may have great therapeutic value in the treatment of MS and other Th1-associated inflammatory disorders.

L13 ANSWER 19 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 11
AN 97021306 EMBASE
DN 1997021306
TI Prevention of a Th1 disease by a Th1 cytokine: **IL-12** and diabetes in NOD mice.
AU **O'Hara R.M. Jr.**; Henderson S.L.; Nagelin A.
CS R.M. O'Hara Jr., Genetics Institute, Laboratory of Molecular Immunology,
87 Cambridge Park Drive, Cambridge, MA 02140, United States
SO Annals of the New York Academy of Sciences, (1996) 795/-(241-249).
Refs: 27
ISSN: 0077-8923 CODEN: ANYAA
CY United States
DT Journal; Conference Article
FS 003 Endocrinology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
AB The effects of interleukin-12 on autoimmune diabetes in nonobese diabetic
mice was examined. **IL-12** was given, intraperitoneally,
to NOD females in two different treatment protocols: three times a week,
for 2 weeks beginning at 9 weeks of age and a single weekly injection, for
15 weeks, beginning at 9 weeks of age. A significant decrease in diabetes
incidence was observed with multidose/short-term **IL-12**
treatment. Age of disease onset, however, was unchanged. Weekly
administration of **IL-12** was more effective in
preventing onset of diabetes. Only 20% of female NOD mice become diabetic
by 30 weeks of age, with a later age of onset. In spite of the decrease in
diabetes incidence, no differences were seen in islet histology with
treated mice compared to controls. Furthermore, **IL-12**
treatment of recipient mice did not prevent induction of diabetes using
spleen cells from diabetic mice in adoptive transfer experiments. These
observations are in contrast to reported data in which treatment of NOD
mice with daily doses of **IL-12** exacerbated disease
incidence and hastened diabetes onset.

L13 ANSWER 20 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 12
AN 97021304 EMBASE

DN 1997021304
TI Regulation of experimental autoimmune encephalomyelitis by interleukin-12.
AU **Leonard J.P.**; Waldburger K.E.; Goldman S.J.
CS J.P. Leonard, Preclinical Biology, Genetics Institute, 1 Burtt Road,
Andover, MA 01810, United States
SO Annals of the New York Academy of Sciences, (1996) 795/- (216-226).
Refs: 28
ISSN: 0077-8923 CODEN: ANYAA
CY United States
DT Journal; Conference Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
LA English
SL English
AB We have evaluated the effects of rmIL-12 on the course of adoptively
transferred EAE. When mice were injected with LNC that had been stimulated
in vitro with PLP in the presence of rmIL-12, the subsequent course of
disease was more severe and prolonged than controls. In vitro stimulation
with PLP in the presence of **IL-12** was associated with
an increase in IFN-.gamma. and decrease in IL-4-producing cells,
indicating a preferential expansion of Th1 effector cells. At peak
disease, no notable differences in either the cellular composition or
cytokine expression within CNS lesions was seen between groups. However,
the frequency of macrophages that stained positively for inducible nitric
oxide synthase (iNOS) was increased in animals challenged with
rmIL-12-treated LNC. These data suggest that in addition to promoting the
preferential expansion of IFN-.gamma.-producing cells by rmIL-12 treatment
in vitro, in vivo effects leading to macrophage activation and iNOS
expression may contribute to the severe, protracted course of CNS
inflammation in this model. In contrast, treatment of mice with an
antibody to murine **IL-12** following cell transfer
completely prevented paralysis with only 40% of the mice developing mild
disease. These data suggest that endogenous **IL-12**
plays a pivotal role in the pathogenesis of this model of autoimmune
disease.

L13 ANSWER 21 OF 24 CAPLUS COPYRIGHT 2002 ACS
AN 1997:285388 CAPLUS
DN 126:329093
TI Effects of interleukin 12 on hematopoietic stem and progenitor cells

AU Neben, Steven; Leonard, John; **Goldman, Samuel**; Ploemacher, Rob E.
CS Department of Immunology and Hematopoiesis, Genetics Institute, Inc., Cambridge, MA, USA
SO Bone Marrow Transplant.: Basic Clin. Stud., [Pap. Int. Symp. BMT] (1996), Meeting Date 1995, 28-35. Editor(s): Ikehara, Susumu; Takaku, Fumimaro; Good, Robert A. Publisher: Springer, Tokyo, Japan.
CODEN: 64HVAW
DT Conference; General Review
LA English
AB A review with 34 refs. Interleukin-12 (**IL-12**) has been shown to possess potent immunomodulatory activity. It has a unique structure among cytokines, consisting of two covalently linked subunits, one with homol. to other members of the cytokine superfamily, the other being highly homologous to gp130, the signaling subunit of a no. of cytokine receptors. Here we summarize studies showing that **IL-12** is a hematopoietic growth factor with potent activity on hematopoietic stem and progenitor cells. In clonal and liq. culture assays, **IL-12** synergizes with IL-3 and Steel Factor to increase the no. of colonies as well as to expand both stem and progenitor cell content in the cultures. In stroma-dependent long-term bone marrow cultures, **IL-12** addn. causes a decrease in cell prodn. in the first week after inoculation of whole bone marrow cells, followed by an increase in both mature cells and progenitor cells over the next 3 wk. The initial decrease appears to be mediated by **IL-12**-induced prodn. of IFN-.gamma., possibly by natural killer cells and/or T cells which do not persist in these cultures. Studies in naive mice demonstrate a similar acute decrease in peripheral leukocyte count, mediated by IFN-.gamma., upon administration of **IL-12**. In contrast, despite a significant decrease in peripheral platelet count, reticulated platelets become elevated and mean megakaryocyte ploidy in the bone marrow shifts from 16N to 32N during **IL-12** treatment. These **IL-12**-mediated effects on megakaryopoiesis are abrogated by simultaneous treatment of mice with antibodies against IFN-.gamma.. These studies provide further information on the potential physiol. role and applications of **IL-12** outside the immune system.

L13 ANSWER 22 OF 24 CAPLUS COPYRIGHT 2002 ACS
AN 1995:934127 CAPLUS

DN 123:337469
TI Use of **IL-12** and **IL-12** antagonists in treatment of autoimmune diseases
IN **Leonard, John P.; Goldman, Samuel; O'Hara, Richard, Jr.**
PA Genetics Institute, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09510444	T2	19971021	JP 1995-524044	19950307
	US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		

AB Autoimmune conditions such as multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or TNF-.alpha., are treated in mammals by administering **IL-12** or an **IL-12** antagonist. Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with **IL-12** during restimulation, and alleviated by injection of a polyclonal antibody to **IL-12**.

L13 ANSWER 23 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 13
AN 95009392 EMBASE
DN 1995009392
TI Prevention of experimental autoimmune encephalomyelitis by antibodies against interleukin 12.
AU **Leonard J.P.**; Waldburger K.E.; Goldman S.J.

CS Preclinical Biology, Genetics Institute, 87 Cambridge Park Drive,Cambridge, MA 02140, United States
SO Journal of Experimental Medicine, (1995) 181/1 (381-386).
ISSN: 0022-1007 CODEN: JEMEAV
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
LA English
SL English
AB Experimental allergic encephalomyelitis (EAE) is an autoimmune disease of the central nervous system that can be transferred to naive mice via CD4+ T cells isolated from appropriately immunized mice. We have evaluated the effects of recombinant murine interleukin 12 (rmIL-12), a potent inducer of interferon .gamma. (IFN-.gamma.) and promoter of Th1 T cell development, on the course of adoptively transferred EAE. The transfer of lymph node cells (LNC) isolated from proteolipid protein (PLP)-primed animals and stimulated in vitro with PLP to naive mice resulted in a progressive paralytic disease culminating in complete hind limb paralysis in the majority of the recipients. When mice were injected with LNC that had been stimulated in vitro with PLP in the presence of rmIL-12, the subsequent course of disease was more severe and prolonged. The addition of rmIL-12 during the in vitro stimulation with PLP resulted in a 10-fold increase in IFN-.gamma. and a 2-fold increase in tumor necrosis factor (TNF) .alpha. in the supernatants, relative to LNC stimulated with PLP alone. However, neutralization of IFN-.gamma. or TNF-.alpha. in vitro with specific antibodies did not abrogate the ability of rmIL-12 to exacerbate the subsequent disease. Similarly, mice treated with rmIL-12 in vivo after the transfer of antigen-stimulated LNC developed a more severe and prolonged course of disease compared with vehicle-treated control animals. In contrast, treatment of mice with an antibody to murine **IL-12** after cell transfer completely prevented paralysis, with only 40% of the mice developing mild disease. These results demonstrate that in vitro stimulation of antigen primed LNC with PLP and rmIL-12 enhances their subsequent encephalitogenicity. Furthermore, inhibition of endogenous **IL-12** in vivo after LNC transfer prevented paralysis, suggesting that endogenous **IL-12** plays a pivotal role in the pathogenesis of this model of autoimmune disease.

L13 ANSWER 24 OF 24 BIOSIS COPYRIGHT 2002 BIOSIS DUPLICATE 14
AN 1993:343704 BIOSIS
DN PREV199396040704
TI Resolution of cutaneous leishmaniasis: Interleukin 12 initiates a protective T helper type 1 immune response.
AU Sypek, Joseph P. (1); Chung, Charles L.; Mayor, Sharon E. H.; Subramanyam, Janaki M.; **Goldman, Samuel L.**; Sieburth, Derek S.; Wolf, Stanley F.; Schaub, Robert G.
CS (1) Dep. Preclin. Biol., Genetics Inst. Inc., 87 Cambridge Park Dr., Cambridge, MA 02140 USA
SO Journal of Experimental Medicine, (1993) Vol. 177, No. 6, pp. 1797-1802.
ISSN: 0022-1007.
DT Article
LA English
AB Resistance to Leishmania major in mice is associated with the appearance of distinct T helper type 1 (Th1) and Th2 subsets. T cells from lymph nodes draining cutaneous lesions of resistant mice are primarily interferon y (IFN-gamma)-producing Th1 cells. In contrast, T cells from susceptible mice are principally Th2 cells that generate interleukin 4 (IL-4). Although existing evidence is supportive of a role for IFN-gamma in the generation of Th1 cells, additional factors may be required for a protective response to be maintained. A potential candidate is **IL-12**, a heterodimeric cytokine produced by monocytes and B cells that has multiple effects on T and natural killer cell function, including inducing IFN-gamma production. Using an experimental leishmanial model we have observed that daily intraperitoneal administration at the time of parasite challenge of either 0.33 mu-g **IL-12** (a consecutive 5 d/wk for 5 wk) or 1.0 mu-g **IL-12** per mouse (only a consecutive 5 d) caused a gt 75% reduction in parasite burden at the site of infection, in highly susceptible BALB/c mice. Delay of treatment by 1 wk had less of a protective effect. Concomitant with these protective effects was an increase in IFN-gamma and a decrease in IL-4 production, as measured by enzyme-linked immunosorbent assay of supernatants generated from popliteal lymph node cells stimulated with leishmanial antigen in vitro. The reduction in parasite numbers induced by **IL-12** therapy was still apparent at 10 wk postinfection. In addition, we observed that the administration of a rabbit anti-recombinant murine **IL-12** polyclonal antibody (200 mu-g i.p. every other day for 25 d) at the time of infection to resistant

C57Bl/6 mice exacerbated disease. These effects were accompanied by a shift in IFN-gamma production in vitro by antigen-stimulated lymph node cells indicative of a Th2-like response. These findings suggest that **IL-12** has an important role in initiating a Th1 response and protective immunity.

=> s arthritis and (il-12 or nksf or clmf)

L14 1030 ARTHRITIS AND (IL-12 OR NKSF OR CLMF)

=> s l14 and tnf

L15 492 L14 AND TNF

=> s l15 and (antagonist? or antibod?)

L16 345 L15 AND (ANTAGONIST? OR ANTIBOD?)

=> dup rem l16

PROCESSING COMPLETED FOR L16
L17 301 DUP REM L16 (44 DUPLICATES REMOVED)

=> s l17 and (therapy or treatment)

4 FILES SEARCHED...
L18 281 L17 AND (THERAPY OR TREATMENT)

=> d bib ab 250-281

L18 ANSWER 250 OF 281 USPATFULL
AN 1998:25211 USPATFULL
TI Cytokine regulatory agents and methods of use in pathologies and conditions associated with altered cytokine levels
IN Girten, Beverly E., San Diego, CA, United States
Andalibi, Ali, San Diego, CA, United States
Basu, Amaresh, San Diego, CA, United States
Fagan, Patrick, Escondido, CA, United States
Houghten, Richard A., Del Mar, CA, United States
Loullis, Costas C., Cardiff, CA, United States
Omholt, Paul, San Diego, CA, United States
Tuttle, Ronald R., Escondido, CA, United States
Suto, Mark J., San Diego, CA, United States
Weber, Patricia A., Stevensville, MT, United States
PA Trega Biosciences, Inc., San Diego, CA, United States (U.S. corporation)
PI US 5726156 19980310
AI US 1995-527056 19950912 (8)
RLI Continuation-in-part of Ser. No. US 1995-484262, filed on 7 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. US 1995-400983, filed on 6 Mar 1995
DT Utility

FS Granted
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner: Delacroix-Muirheid, C.
LREP Campbell & Flores LLP
CLMN Number of Claims: 31
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1873
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel peptides that are potent cytokine regulatory agents. In addition, the present invention relates to pharmaceutical compositions comprising a pharmaceutically acceptable carrier and a cytokine regulatory agent. Administration of such a cytokine regulatory agent to a subject can enhance or restrain cytokine activity. Thus, the present invention provides a method of regulating cytokine activity in a subject having a condition characterized by aberrant or altered cytokine activity. The invention also provides methods of treating such conditions, including, for example, disuse deconditioning, diseases mediated by nitric oxide and cytokines, adverse drug reactions, obesity, septic shock, and adverse side effects due to cancer chemotherapy or occurring as in response to organ transplantation.

L18 ANSWER 251 OF 281 USPATFULL
AN 1998:22079 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5723315 19980303
AI US 1996-702344 19960823 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2437
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L18 ANSWER 252 OF 281 USPATFULL
AN 1998:4755 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewskbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Brookline, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5708157 19980113
AI US 1996-686878 19960726 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3204
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L18 ANSWER 253 OF 281 USPATFULL
AN 1998:4432 USPATFULL
TI DNA sequences and secreted proteins encoded thereby
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
McCoy, John M., Reading, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5707829 19980113
AI US 1995-514014 19950811 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Nashed, Nashaat T.
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 44
ECL Exemplary Claim: 44
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1689
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L18 ANSWER 254 OF 281 USPATFULL
AN 1998:1445 USPATFULL
TI Gene **therapy** for T cell regulation
IN Dow, Steve W., Denver, CO, United States
Elmslie, Robyn E., Denver, CO, United States
PA National Jewish Center for Immunology & Respiratory Medicine, Denver,

CO, United States (U.S. corporation)
PI US 5705151 19980106
AI US 1995-446918 19950518 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Chambers, Jasemine C.; Assistant Examiner: Hauda,
Karen M.
LREP Sheridan Ross P.C.
CLMN Number of Claims: 52
ECL Exemplary Claim: 1,28
DRWN 10 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2206
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a nucleic acid-based therapeutic composition to treat an animal with disease by controlling the activity of effector cells, including T cells, macrophages, monocytes and/or natural killer cells, in the animal. The present invention also relates to methods of gene **therapy** involving different modes of administration of a therapeutic composition to treat animals with different types of diseases. Also included in the present invention are recombinant molecules for use in a therapeutic composition and recombinant cells useful as a tumor vaccine. Therapeutic compositions of the present invention include superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule, depending upon the disease being treated.

L18 ANSWER 255 OF 281 USPATFULL
AN 97:117939 USPATFULL
TI Methods and compositions for inhibiting production of replication competent virus
IN Klump, Wolfgang M., Del Mar, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
PA Chiron Corporation, United States (U.S. corporation)
PI US 5698446 19971216
AI US 1994-305699 19940907 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Guzo, David
LREP Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2090
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for inhibiting the production of replication competent virus. The invention comprises

nucleic acid cassettes encoding a non-biologically active inhibitory molecule which are incorporated into packaging cells and recombinant vector constructs. Upon recombination between various vector construct contained within the producer cell, a biologically active molecule is produced which kills the cell, thereby inhibiting production of replication competent virus.

L18 ANSWER 256 OF 281 USPATFULL
AN 97:117893 USPATFULL
TI Detecting genetic predisposition for osteoporosis
IN Duff, Gordon W., 18 Ashgate Road, Sheffield, S10 3BZ, S Yorks, England
Russell, Graham, Ronksley Farm Hollow Meadows, Sheffield, South Yorks S6 6GH, England
Eastell, Richard, 289 Ringinglow Road, Sheffield, S11 7PZ, England
PI US 5698399 19971216
AI US 1996-628282 19960405 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Horlick, Kenneth R.
LREP Jenkens & Gilchrist
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 668
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods of predicting the risk of osteoporosis. Specifically, the methods comprise isolating genomic DNA from an individual and determining an allelic pattern for IL-1 receptor **antagonist** (IL-1ra) in the genomic DNA. The identification of at least one copy of allele 2 indicates increased susceptibility to osteoporosis.

L18 ANSWER 257 OF 281 USPATFULL
AN 97:81140 USPATFULL
TI DNA encoding natural killer lytic associated protein
IN Kornbluth, Jackie, 174 Pebble Beach Dr., Little Rock, AR, United States 72212
PI US 5665588 19970909
AI US 1995-398008 19950302 (8)
RLI Continuation-in-part of Ser. No. US 1993-126501, filed on 24 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ziska, Suzanne E.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1

DRWN 18 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 1252
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A unique gene sequence encoding a natural killer lytic associated molecule (natural killerlytic associated protein) has been isolated.

Using recombinant DNA techniques, the natural killerlyric associated protein may be produced in useful quantities. Methods for preparing the gene sequence and the gene product are disclosed, as well as methods of using the product to enhance anti-tumor, anti-viral and anti-microbial activity of natural killer cells. A method of inhibiting expression of the gene product is also disclosed, which may be used to turn off immune responses in situations of graft rejection and autoimmune disorders.

Furthermore, methods of treating tumors and viruses in humans have been developed.

L18 ANSWER 258 OF 281 USPATFULL
AN 97:80900 USPATFULL
TI **IL-12** inhibition of B1 cell activity
IN Metzger, Dennis W., Sylvania, OH, United States
Van Cleave, Victor H., Londonderry, NH, United States
PA Genetics Institute, Cambridge, MA, United States (U.S. corporation)
Medical College of Ohio, Toledo, OH, United States (U.S. corporation)
PI US 5665347 19970909
AI US 1995-382658 19950202 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Achutamurthy, Ponnathapura
LREP Hamilton, Brook, Smith & Reynolds, P.C.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1,2
DRWN 47 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 942
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to a method of suppressing B1 cell activity in a host (e.g., mammalian, including human) comprising administering to the host an effective amount of **IL-12** that significantly suppresses or inhibits B1 cell activity. In addition, the invention relates to a method of treating a B1 cell disorder in a host, comprising administering to the host an effective therapeutic amount of **IL-12**. The invention further encompasses a method of screening for substances (e.g., proteins, peptides, small molecules) which enhance or suppress the inhibition of B1 cell activity by **IL-**

12. The invention also relates to a substance identified by the methods of screening for a substance which enhances or suppresses
IL-12 inhibition of B1 cell activity.

L18 ANSWER 259 OF 281 USPATFULL
AN 97:68346 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5654173 19970805
AI US 1996-702080 19960823 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jagannathan, Vasu S.; Assistant Examiner: Lathrop,
Brian
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1685
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L18 ANSWER 260 OF 281 USPATFULL
AN 97:64091 USPATFULL
TI P-40 homodimer of interleukin-12
IN Gately, Maurice Kent, Pine Brook, NJ, United States
Hakimi, John, Scarsdale, NY, United States
Ling, Ping, Nutley, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5650492 19970722
AI US 1995-424682 19950418 (8)
RLI Continuation of Ser. No. US 1993-87832, filed on 2 Jul 1993, now
abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John; Assistant Examiner: Mertz, Prema
LREP Johnston, George W., Tramaloni, Dennis P., Kass, Alan P.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analysis of the culture media of p40-transfected COS cells indicated the
presence of 40 kDa monomers and 80 kDa disulfide-linked homodimers.

Examination of partially purified p40 recombinant proteins demonstrated that only the homodimer but not the monomer binds to the **IL-12** receptor. Partially purified 80 kDa homodimer inhibited [.sup.125 I]**IL-12** binding to PHA-activated human lymphoblasts with an IC.sub.50 of 80 ng/ml, which is similar to the IC.sub.50 value (20 ng/ml) for the human **IL-12** heterodimer. Although neither the 40 kDa monomer nor the 80 kDa dimer could stimulate human PHA-blast proliferation, the 80 kDa dimer inhibited **IL-12**-induced proliferation in a dose-dependent manner with an IC.sub.50 of 1 .mu.g/ml. The **IL-12** p40 subunit contains the essential epitopes for receptor binding, but they are only active when p40 is covalently associated with a second protein such as p35 or p40. When p40 is associated with the p35 subunit, the heterodimer acts as an agonist mediating biologic activity. When p40 associates with itself, the homodimer behaves as an **antagonist**.

L18 ANSWER 261 OF 281 USPATFULL
AN 97:54233 USPATFULL
TI Substituted amino alcohol compounds
IN Klein, J. Peter, Vashon, WA, United States
Underiner, Gail E., Brier, WA, United States
Kumar, Anil M., Seattle, WA, United States
PA Cell Therapeutics, Inc., Seattle, WA, United States (U.S. corporation)
PI US 5641783 19970624
AI US 1994-303842 19940908 (8)
RLI Continuation-in-part of Ser. No. US 1993-152650, filed on 12 Nov 1993
And Ser. No. US 1993-164081, filed on 8 Dec 1993, now patented, Pat. No.
US 5470878
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Cebulak, Mary
C.
LREP Faciszewski, Stephen, Oster, Jeffrey B.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 115 Drawing Figure(s); 88 Drawing Page(s)
LN.CNT 3206
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are compounds having a straight or branched aliphatic hydrocarbon structure of formula I: ##STR1## In formula I, n is an integer from one to four and m is an integer from four to twenty.
Independently, R.sub.1 and R.sub.2 are hydrogen, a straight or branched chain alkyl, alkenyl or alkynyl of up to twenty carbon atoms in length or --(CH.sub.2).sub.w R.sub.5. If R.sub.1 or R.sub.2 is

--(CH.sub.2).sub.w R.sub.5, w may be an integer from one to twenty and
R.sub.5 may be an hydroxyl, halo, C.sub.1-8 alkoxyl group or a substituted or unsubstituted carbocycle or heterocycle. Alternatively,
R.sub.1 and R.sub.2 may jointly form a substituted or unsubstituted,
saturated or unsaturated heterocycle having from four to eight carbon
atoms, N being a hetero atom of the resulting heterocycle. R.sub.3 may
be either hydrogen or C.sub.1-3. In the compounds, a total sum of carbon
atoms comprising R.sub.1 or R.sub.2, (CH.sub.2).sub.n and
(CH.sub.2).sub.m does not exceed forty. R.sub.4 is a terminal moiety
comprising a substituted or unsubstituted, oxidized or reduced ring
system, the ring system having a single ring or two to three fused
rings, a ring comprising from three to seven ring atoms. The disclosed
compounds are effective agents to inhibit undesirable responses to cell
stimuli.

L18 ANSWER 262 OF 281 USPATFULL
AN 97:25018 USPATFULL
TI Method of making inosine monophosphate derivatives and immunopotentiating uses thereof
IN Hadden, John W., Tampa, FL, United States
Giner-Sorolla, Alfredo, Tampa, FL, United States
PA The University of South Florida, Tampa, FL, United States (U.S. corporation)
PI US 5614504 19970325
AI US 1995-426682 19950421 (8)
RLI Continuation-in-part of Ser. No. US 1992-995550, filed on 22 Dec 1992,
now abandoned which is a continuation of Ser. No. US 1990-561979, filed
on 1 Aug 1990, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Kight, John; Assistant Examiner: Crane, L. Eric
LREP Kohn & Associates
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 19 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 2194
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of making inosine-5'-monophosphate and its derivatives resistant to 5'-nucleotidase by chemically modifying
inosine-5'-monophosphate to the formula: ##STR1## wherein R is selected
from the group consisting of an alkyl, alkoxy and secondary amino
compounds whereby inosine-5'-monophosphate biological activity is

retained in vitro and extended to in vivo.

L18 ANSWER 263 OF 281 USPATFULL
AN 97:3820 USPATFULL
TI Genetic immunization
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
PI US 5593972 19970114
AI US 1993-125012 19930921 (8)
RLI Continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993,
now abandoned which is a continuation-in-part of Ser. No. US 1993-8342,
filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Fleisher, Mindy; Assistant Examiner: Railey, II,
Johnny F.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 3611
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of prophylactic and therapeutic immunization of an individual
against pathogen infection, diseases associated with hyperproliferative
cells and autoimmune diseases are disclosed. The methods comprise the
steps of administering to cells of an individual, a nucleic acid
molecule that comprises a nucleotide sequence that encodes a protein
which comprises at least one epitope that is identical or substantially
similar to an epitope of a pathogen antigen, a hyperproliferative cell
associated protein or a protein associated with autoimmune disease
respectively. In each case, nucleotide sequence is operably linked to
regulatory sequences to enable expression in the cells. The nucleic acid
molecule is free of viral particles and capable of being expressed in
said cells. The cells may be contacted cells with a cell stimulating
agent. Methods of prophylactically and therapeutically immunizing an
individual against HIV are disclosed. Pharmaceutical compositions and

kits for practicing methods of the present invention are disclosed.

L18 ANSWER 264 OF 281 USPATFULL
AN 96:120774 USPATFULL
TI Tetracycline regulated transcriptional modulators with altered DNA
binding specificities
IN Bujard, Hermann, Heidelberg, Germany, Federal Republic of
Gossen, Manfred, El Cerrito, Germany, Federal Republic of
Hillen, Wolfgang, Erlangen, Germany, Federal Republic of
Helbl, Vera, Fuerth, Germany, Federal Republic of
Schnappinger, Dirk, Bad Driburg, Germany, Federal Republic of
PA BASF Aktiengesellschaft, Ludwigshafen, Germany, Federal Republic of
(non-U.S. corporation)
Knoll Aktiengesellschaft, Ludwigshafen, Germany, Federal Republic of
(non-U.S. corporation)
PI US 5589362 19961231
AI US 1995-485971 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-383754, filed on 3 Feb 1995 And
a continuation-in-part of Ser. No. US 1994-275876, filed on 15 Jul 1994
And a continuation-in-part of Ser. No. US 1994-260452, filed on 14 Jun
1994 And a continuation-in-part of Ser. No. US 1993-76726, filed on 14
Jun 1993, now patented, Pat. No. US 5464758 , said Ser. No. US -275876
which is a continuation-in-part of Ser. No. US 1994-270637, filed on 1
Jul 1994, now abandoned , said Ser. No. US -260452 which is a
continuation-in-part of Ser. No. US 1993-76327, filed on 14 Jun 1993,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Elliott, George C.; Assistant Examiner: Brusca, John
S.
LREP Lahive & Cockfield, DeConti, Jr., Giulio A.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 4415
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Isolated nucleic acid molecules encoding fusion proteins which regulate
transcription in eukaryotic cells are disclosed. The fusion proteins of
the invention comprise a Tet repressor having at least one amino acid
mutation that confers on the fusion protein an ability to bind a class B
tet operator sequence having a nucleotide substitution at position +4 or
+6, operatively linked to a polypeptide which regulates transcription in

eukaryotic cells. Methods for regulating transcription of a tet operator-linked gene in a cell are also provided. In one embodiment, the method involves introducing into the cell a nucleic acid molecule encoding a fusion protein which regulates transcription, the fusion protein comprising a Tet repressor having at least one amino acid mutation that confers on the fusion protein an ability to bind a class B tet operator sequence having a nucleotide substitution at position +4 or +6, operatively linked to a polypeptide which regulates transcription in eukaryotic cells, and modulating the concentration of a tetracycline, or analogue thereof, in contact with the cell.

L18 ANSWER 265 OF 281 USPATFULL
AN 96:113801 USPATFULL
TI Evaluation and **treatment** of patients with progressive immunosuppression
IN Ochoa, Augusto C., Washington, DC, United States
Mizuguchi, Hiromoto, Frederick, MD, United States
O'Shea, John J., Silver Spring, MD, United States
Longo, Dan L., Kensington, MD, United States
Loeffler, Cynthia M., Pensacola, FL, United States
PA Regents of the University of Minnesota, Minneapolis, MN, United States
(U.S. corporation)
The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5583002 19961210
AI US 1992-987966 19921211 (7)
RLI Continuation of Ser. No. US 1992-863262, filed on 6 Apr 1992, now patented, Pat. No. US 5296353
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2252
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A soluble immunosuppressive factor present in serum derived from tumor-bearing mammals, is associated with changes in TCR protein subunit levels and T-lymphocyte signal transduction pathway proteins. These changes provide a method of determining the level of immunosuppression in a mammal by determining the level of expression of at least one selected TCR subunit protein, or a protein in the T lymphocyte signal

transduction pathway, and comparing the level to that found in non-immunosuppressed individuals. The method is useful to identify patients having T lymphocytes capable of activation for immunotherapy and for identifying agents which cause or reverse immunosuppression. An isolated immunosuppressive factor associated with the level of expression of the proteins is useful for suppressing the immune response, for example, in organ transplantation.

L18 ANSWER 266 OF 281 USPATFULL
AN 96:85044 USPATFULL
TI Evaluation and **treatment** of patients with progressive immunosuppression
IN Ochoa, Augusto C., Frederick, MD, United States
Longo, Dan L., Kensington, MD, United States
Ghosh, Paritosh, Frederick, MD, United States
Young, Howard A., Geithersburg, MD, United States
PA United States of America as represented by the Secretary of the Department of Health and Human Services, Washington, DC, United States
(U.S. government)
PI US 5556763 19960917
AI US 1993-34832 19930317 (8)
RLI Continuation-in-part of Ser. No. US 1993-31434, filed on 15 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-987966, filed on 11 Dec 1992 which is a continuation-in-part of Ser. No. US 1992-863262, filed on 6 Apr 1992, now patented, Pat. No. US 5296353
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Foley & Lardner
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2646
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A soluble immunosuppressive factor present in serum derived from tumor-bearing mammals, is associated with changes in TCR protein subunit levels, T lymphocyte signal transduction pathway proteins. These changes provide a method of determining the level of immunosuppression in a mammal by determining the level of expression of at least one selected TCR subunit protein, a protein in the T lymphocyte signal transduction pathway, or of the NF-.kappa.B/rel family and comparing the level and pattern to that found in non-immunosuppressed individuals. The method is useful to identify patients having T lymphocytes capable of activation

for immunotherapy and for identifying agents which cause or reverse
immunosuppression. An isolated immunosuppressive factor associated with
the level of expression of the proteins is useful for suppressing the
immune response, for example, in organ transplantation.

L18 ANSWER 267 OF 281 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor
cDNAs and protein and uses therefore. The Interleukin-12 receptor is
shown to be a member of the cytokine receptor superfamily and has a high
homology to human gp130.

L18 ANSWER 268 OF 281 USPATFULL
AN 94:24192 USPATFULL
TI Evaluation and **treatment** of patients with progessive immunosuppression
IN Ochoa, Augusto C., Washington, DC, United States
Mizoguchi, Hiromoto, Frederick, MD, United States
O'Shea, John J., Silver Spring, MD, United States
Longo, Dan L., Kensington, MD, United States
Loeffler, Cynthia M., Bladensburg, MD, United States
PA The United States of America as represented by the Department of Health
and Human Services, Washington, DC, United States (U.S. government)
Regents of the University of Minnesota, Minneapolis, MN, United States
(U.S. corporation)
PI US 5296353 19940322
AI US 1992-863262 19920406 (7)
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Foley & Lardner

CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1393
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of determining the level of immunosuppression in a mammal
involves determining the level of expression of at least one selected
TCR subunit protein, or protein in the T lymphocyte signal transduction
pathway, and comparing the level to that found in healthy individuals.
The method is useful to identify patients having T lymphocytes capable
of activation for autologous adoptive immunotherapy and for identifying
agents which cause or reverse immunosuppression.

L18 ANSWER 269 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-648241 [74] WPIDS
DNC C2001-191222
TI N-Aryl 4-(optionally fused heteroaryl)-2-thiazolamines are **TNF** and IL cytokine inhibitors, useful for inflammatory and autoimmune disorders, e.g. **arthritis**, irritable bowel, transplants, asthma and shock.
DC B02 B03
IN COOYMANS, L; DE BRABANDER, M; KENNIS, L E J; LOVE, C; VAN WAUWE, J P F;
VANDERMAESEN, N
PA (JANC) JANSSEN PHARM NV
CYC 94
PI WO 2001064674 A1 20010907 (200174)* EN 99p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001037401 A 20010912 (200204)
ADT WO 2001064674 A1 WO 2001-EP1841 20010220; AU 2001037401 A AU 2001-37401
20010220
FDT AU 2001037401 A Based on WO 200164674
PRAI EP 2000-200733 20000301
AB WO 200164674 A UPAB: 20011217
NOVELTY - Use of N-aryl 4-(optionally fused heteroaryl)-2-thiazolamines
(I), or their N-oxides, simple and quaternary salts, and stereoisomers,
for **treatment** and prophylaxis of cytokine mediated diseases.
DETAILED DESCRIPTION - Use of N-aryl 4-(optionally fused heteroaryl)-2-thiazolamines of formula (I), or their N-oxides, simple and

quaternary salts, and stereoisomers, for **treatment** and prophylaxis of cytokine mediated diseases, is new.

Q = 3-6C cycloalkyl, phenyl, naphthyl, pyridyl, pyrimidinyl, pyrazinyl, pyridazinyl, benzothiazolyl, benzoxazolyl, benzimidazolyl, indazolyl, or imidazolyl (all optionally substituted by 1-3 J), or a hetero-fused phenyl group (a), (b), or (c):

J = halogen, hydroxy, cyano, carboxy, azido, amino, mono- or di- (1-6C alkyl)amino, 1-6C alkyl (optionally substituted), alkoxy, or alkylthio, 2-6C alkenyl or alkynyl, 2-7C alkylcarbonyl or alkoxycarbonyl, aryloxy, aryl 1-6C alkoxy, 1-4C alkylsulfinyl or alkylsulfonyl, or 1-4C alkylaminosulfinyl, alkylaminosulfonyl or R1HN-S(=O)n-;

n = 0, 1 or 2;

X, Y = O, NR3, CH2, or S;

Z' = O or NR4;

q = 1-4;

r = 1-3;

L = phenyl or Het (both optionally substituted by 1-4 G, or 1-6 G for fused bicyclic Het);

G = halogen, hydroxy, amino, cyano, carboxy, mono- or di- (1-6C alkyl)amino, 1-6C alkyl (optionally substituted) or alkoxy, or 2-7C alkylcarbonyl, alkylcarbonyloxy, alkylcarbonylamino, or alkoxycarbonylamino, aminocarbonyl, or mono- or di- (1-6C alkyl)aminocarbonyl;

aryl = phenyl (optionally substituted by 1-5 of halo, hydroxy, (polyhalo) 1-6C alkyl, 1-6C alkyloxy, 1-6C alkylthio, cyano, nitro, amino or mono- or di-(1-6C alkyl)amino);

R1 = H, or an azacyclic group (d):

R2a = H, or 1-6C alkyl or alkoxy;

A = O, S, or CR2a=N with the C attached to the NH; and

Het = 5 or 6 membered heterocyclyl containing 1-4 heteroatoms from N, O, S and at least 2 double bonds, optionally fused through C or N atoms to a 5 or 6 membered saturated, partially unsaturated, or aromatic, otherwise carbocyclic or heterocyclic ring.

INDEPENDENT CLAIMS are also included for:

(1) the compounds of formula (I) with provisos. The provisos are listed in FULL DEFINITIONS in the DEFINITIONS FIELD;

(2) several preparations of compound (I); and

(3) a composition comprising a new compound of formula (I) and another antiinflammatory or immunosuppressive compound.

ACTIVITY - Antiinflammatory; antiarthritic; antiallergic; antiparasitic; antimalarial; antidiabetic; antiasthmatic; immunosuppressive; hepatotropic; nephrotrophic; vasotropic; tuberculostatic; vulnerary; antiparkinsonian; antithyroid;

immunomodulator; antiviral; antirheumatic; dermatological; ophthalmological; antibacterial; antiparasitic; antipsoriatic; antiparkinsonian; antipyretic.

MECHANISM OF ACTION - (I) are inhibitors and/or **antagonists** of proinflammatory cytokines, notably **TNF**-alpha and/or **IL-12**. They also have selective affinity for, and block, the adenosine A3 receptor. Tests were conducted with cell free human peripheral blood to determine inhibition of **TNF**- alpha and **IL-12** by compounds (I) at a concentration of 100 nM. Respective results for a range of compounds were 39-56%, and 53-75% with one 86%.

USE - For **treatment** or prevention of diseases mediated through activation of the adenosine A3 receptor (claimed). For use in the prevention and **treatment** of inflammatory or autoimmune disorders (such as rheumatoid **arthritis**, Crohn's disease, irritable bowel disease and colitis) (claimed). For **treatment** or prevention of diseases mediated through cytokines (specifically Tumor Necrosis Factor-alpha (**TNF**- alpha) and Interleukin 12 (**IL-12**) mediated diseases) (claimed).

For **treatment** of rheumatoid spondylitis, spondyloarthropathies, systemic lupus erythematosus, **arthritis**, polychondritis, sclerodoma, Wegener granulamatosis, dermatomyositis, Steven-Johnson syndrome, idiopatic sprue, endocrine opthalmopathy, Grave's disease, alveolitis, chronic hypersensitivity pneumonitis, primary billiary cirrhosis, uveitis, keratoconjunctivitis sicca and vernal keratoconjunctivitis, allergic rhinitis, pemphigus, eosinophilia, Loffler's syndrome, eosinophilic pneumonia, parasitic infestation, bronchopulmonary aspergillosis. polyarteritis nodosa, eosinophilic granuloma, eosinophil-related disorders affecting the airways occasioned by drug-reaction, sepsis, septic shock, endotoxic shock, gram negative sepsis, toxic shock syndrome, cerebral malaria, adult respiratory distress syndrome, bronchitis, chronic obstructive airway or pulmonary disease, pulmonary fibrosis, pneumocomosis, tuberculosis, silicosis, exacerbation of airways hyperreactivity to other drug **therapy** (e.g. aspirin or beta -agonist **therapy**), pulmonary sarcoidosis, bone resorption diseases, meningitis, reperfusion injury, graft versus host reaction, allograft rejections, transplant rejections, fever and royalgias due to infection, such as influenza, cachexia, AIDS, ARC (AIDS related complex), diabetes, cancer, angiogenesis, lymphoma, Kawasaki syndrome, Behcet's syndrome, aphthous ulceration, skin-related disorders (such as

psoriasis and eczema), bowel disease (such as Crohn's disease), pyresis,
asthma, wheezy infant syndrome, multiple sclerosis, Parkinson's disease,
pancreatitis, cardiac disease, congestive heart failure, myocardial
infarction, acute liver failure, glomerulonephritis, **therapy**-associated syndromes comprising Jarisch-Herxheimer reaction, and syndromes associated with IL-2 infusion,
anti-CD3 **antibody** infusion, hemodialysis and yellow fever vaccination.
ADVANTAGE - (I) are stated to be more specific and less toxic than
present antiinflammatory and immunosuppressive drugs, and may be used in
combination with them to reduce dosing and side effects.
Dwg.0/0

L18 ANSWER 270 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-596713 [67] WPIDS
DNC C2001-176532
TI Novel conjugate for treating lesions, comprises a specific binding member
specific for extra-cellular matrix component present in lesions, and a
molecule that exerts biocidal/cytotoxic effect on target cells in lesions.
DC B04 D16
IN BORSI, L; CARNEMOLLA, B; HALIN, C; NERI, D; NILSSON, F; TARLI, L; ZARDI, L
PA (PHIL-N) PHILOGEN SRL
CYC 94
PI WO 2001062298 A2 20010830 (200167)* EN 88p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001039470 A 20010903 (200202)
ADT WO 2001062298 A2 WO 2001-IB382 20010222; AU 2001039470 A AU 2001-39470
20010222
FDT AU 2001039470 A Based on WO 200162298
PRAI US 2000-257192P 20001221; US 2000-184767P 20000224
AB WO 200162298 A UPAB: 20011119
NOVELTY - A conjugate (I) of a binding member (II) specific for an
extra-cellular matrix component present in angiogenesis in pathological
lesions, and a molecule (III) which exerts a biocidal or cytotoxic effect
on target cells by cellular interaction, is new.
DETAILED DESCRIPTION - A conjugate (I), comprises a binding member

(II) specific for an extra-cellular matrix component present in angiogenesis in pathological lesions, and a molecule (III) which exerts a biocidal or cytotoxic effect on target cells by cellular interaction. (II) further comprises one or more VH and/or VL domains of **antibody** L19 and/or competes with **antibody** L19 for binding fibronectin ED-B.

In (I), the amino acid sequences of the VH and VL domains of **antibody** L19 is disclosed in Pini et al. (1998) J. Biol. Chem. 273: 21769-21776.

ACTIVITY - Cytostatic; antirheumatic; antiarthritic; antidiabetic; ophthalmological. The efficacy of the L19-IL2 fusion protein was tested on mouse teratocarcinoma, F9, mouse adenocarcinoma, C51 and human small cell lung cancer, N592. Cells of each tumor type were injected subcutaneously into the animals and left for 24 hours. The animals received daily intravenous injections of either phosphate buffered saline (PBS), a mixture of L19 and IL2, or L19-IL2 fusion protein. After 24 hours the animals were sacrificed, the tumoral mass removed and the tumors were weighed. The results showed a significant decrease in tumor growth in the group of animals treated with L19-IL2 fusion protein with respect both to animals injected with an equimolar mixture of L19 and IL2 proteins and to the third group treated with PBS.

MECHANISM OF ACTION - Competes with **antibody** L19 for binding to fibronectin ED-B (claimed). No biological data was provided.

USE - (I) is useful for treating a human or animal by **therapy**, and in the manufacture of medicament for **treatment** of angiogenesis in pathological lesions, and for treating tumors (claimed).

(I) is also useful for treating lesions of pathological angiogenesis such as rheumatoid **arthritis**, diabetic retinopathy, age related muscular degeneration and angiomas.

Dwg.0/23

L18 ANSWER 271 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD

AN 2001-244697 [25] WPIDS

DNC C2001-073427

TI Modulating responsiveness to a corticosteroid by administering a corticosteroid with an agent which antagonizes a target that regulates interferon-gamma production or an caspase family protease inhibitor, useful for treating asthma.

DC B04 B05 D16

IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E
PA (BADI) BASF AG
CYC 94
PI WO 2001019373 A2 20010322 (200125)* EN 152p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000071276 A 20010417 (200140)
ADT WO 2001019373 A2 WO 2000-US24725 20000908; AU 2000071276 A AU 2000-71276
20000908
FDT AU 2000071276 A Based on WO 200119373
PRAI US 1999-398555 19990917
AB WO 200119373 A UPAB: 20010508
NOVELTY - A new method (M1) for modulating responsiveness to a corticosteroid in a subject comprises administering a corticosteroid with an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) or at least one agent (A2) that is an inhibitor of a caspase family protease.

DETAILED DESCRIPTION - A method (M1) for modulating responsiveness to a corticosteroid in a subject, comprising selecting a subject in need of modulation of responsiveness to a corticosteroid and administering:

(a) an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) in the subject, the agent being administered at a dosage and by a route sufficient to inhibit production of IFN-gamma; or

(b) at least one agent (A2) that is an inhibitor of a caspase family protease; and

(c) a corticosteroid.

The responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

An INDEPENDENT CLAIM is also given for a method (M2) for regulating the production of IFN-gamma in a subject, comprising administering a corticosteroid and an agent which antagonizes a target that regulates production of IFN-gamma such that production of IFN-gamma is modulated in the subject.

ACTIVITY - Immunosuppressive; antiinflammatory; dermatological;

antibacterial; cytostatic; antiasthmatic; anticonvulsant; antidiabetic;
antiarthritic; antirheumatic; neuroprotective; antiallergic; antiulcer;
ophthalmological; antianemic.

Interleukin converting enzyme (ICE)-deficient and wild type mice
first were sensitized with Propionibacterium acnes cell wall material (1
mg per mouse) to induce low grade inflammation and six days later were
challenged with lipopolysaccharide (LPS) (1 microgram per mouse in 0.1 ml
of saline intravenously). Thirty minutes after LPS administration, the
mice were treated with the corticosteroid dexamethasone (4 mg/kg per mouse
in 0.5 ml 95% saline/0.5% ethanol, intraperitoneally). Control mice were
treated with vehicle alone. All mice were bled 90 minutes after LPS
administration and the serum samples were analyzed for the presence of
tumor necrosis alpha (**TNF**-alpha) by standard ELISA (Enzyme linked immunosorbant assay).

Wild type and ICE deficient mice treated with vehicle alone had
similar levels of serum **TNF**-alpha. **Treatment** of wild type mice with dexamethasone did not significantly affect serum **TNF**-alpha levels, demonstrating their resistance to steroid **treatment** in this septic shock model. In contrast, **treatment** of the ICE deficient mice with dexamethasone suppressed serum **TNF**-alpha levels by 74% (p less than 0.002). These data indicate that inhibition of ICE activity reverses resistance to steroid **treatment** in a septic shock model.

MECHANISM OF ACTION - **IL-12 antagonist**; IL-18 **antagonist**; phosphodiesterase IV inhibitor; a beta-2 agonist; a STAT4 inhibitor; an anti-IL-1-alpha **antibody**; an anti-IL-1-beta **antibody**; an anti-tumor necrosis factor **antibody**; a natural killer cell **antagonist**; a T-cell **antagonist**; caspase family protease inhibitor; gene **therapy**.

USE - The method is useful for treating a subject suffering from an autoimmune disease or disorder, an acute (e.g. infectious meningitis) or chronic (e.g. systemic lupus erythematosus or psoriasis) inflammatory disorder, septic shock or sepsis, graft versus host disease or transplant rejection, complications associated with post-surgical stress, Still's disease, leukemia or an immuno-inflammatory disease or disorder. The immuno-inflammatory disease or disorder is asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel

disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune **arthritis**, rheumatoid **arthritis**, juvenile rheumatoid **arthritis**, psoriatic **arthritis**, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior or interstitial lung fibrosis (claimed).

The method is useful for modulating corticosteroid responsiveness in a variety of clinical settings, for e.g. reversing steroid resistance, increasing steroid sensitivity, ameliorating a steroid rebound effect associated with administration of reduced dosages of the corticosteroid, or modulating corticosteroid activity, such that the corticosteroids can be tapered to zero (claimed).

Dwg.0/12

L18 ANSWER 272 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-244560 [25] WPIDS
DNC C2001-073385
TI Composition comprising interleukin-12 p40 and IL-B30 polypeptide or its segment, useful for ameliorating rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 91
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ

EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV
MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT
TZ UA UZ VN YU ZA

AU 2000073608 A 20010410 (200137)

ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608
20000908

FDT AU 2000073608 A Based on WO 200118051

PRAI US 1999-164616P 19991110; US 1999-393090 19990909

AB WO 200118051 A UPAB: 20010508

NOVELTY - A composition (I) comprising a substantially pure polypeptide
comprising a number of distinct segments of at least 7 contiguous amino
acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and
a substantially pure polypeptide comprising a segment of at least 11
contiguous amino acids from **IL-12** p40 and/or IL-B30.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:

(1) an isolated or recombinant nucleic acid (II) encoding (I);

(2) a cell (III) comprising (II);

(3) a nucleic acid (IV) which hybridizes under wash conditions of 30
minutes at 50 deg. C and less than 1M salt to the natural mature coding
portion of primate **IL-12** p40 and IL-B30;

(4) an **antagonist** (V) of **IL-12** p40/IL-B30 combined with a tumor necrosis factor-alpha (**TNF** alpha) **antagonist**, an **IL-12** **antagonist**, IL-10, or steroids;

(5) a binding compound (VI) comprising an antigen binding site from
an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12** p40 and IL-B30 polypeptide, or a polypeptide comprising **IL-12** p40 fused to IL-B30, but not to either **IL-12** p40 or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further
comprising a primate **IL-12** p40 or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12** p40/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI)

and a carrier such as an aqueous compound, including water, saline, and/or
buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves
expressing the polypeptide with **IL-12** p40 or
increasing the secretion of a primate **IL-12** p40
involves expressing the **IL-12** p40 with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves
contacting the complex to a cell expressing the receptor under conditions
allowing the complex to bind to the receptor, forming a detectable
interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic;
neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic;
cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell
in host; inducer of memory T-cell proliferation (claimed); modulator of
trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a
cell or tissue in a host organism by contacting the cell with (I) or (V),
resulting in an increased or decreased production of Interferon-gamma (IFN
gamma), an enhanced Th1 response such as anti-tumor effect, adjuvant
effect, anti-viral effect or antagonized allergic effect, and amelioration
of an autoimmune condition or a chronic inflammatory condition. The
contacting is in combination with IL-18, **IL-12**,
radiation **therapy** or chemotherapy, an immune adjuvant or an
anti-viral therapeutic. The **antagonist** is an **antibody**
against **IL-12** receptor subunit beta 1. The
antagonist or agonist of mammalian IL-B30 protein is useful for
modulating the inflammatory response in an animal, by contacting cells in
the animal with the agonist or **antagonist**, where the animal
exhibits signs or symptoms of an acute phase inflammatory response in
skin, lung, gastrointestinal, or liver tissue. The modulation is
accelerating maturation of neutrophils into platelets and has an effect on
immunoglobin A and G (IgA and IgG) . The **antagonist** is an
antibody which binds to the mammalian IL-B30 or blocks signaling
mediated by mammalian IL-B30. The **antagonist** or agonist is
administered in combination with an anti-inflammatory cytokine agonist or
antagonist, an analgesic, an anti-inflammatory agent, or a
steriod. IL-B30 or its agonist is useful inducing the proliferation of
memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

IL-12 p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L18 ANSWER 273 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2000-687263 [67] WPIDS
DNC C2000-209152
TI Treating graft-versus-host disease, cancer, immunodeficiency or an autoimmune disease comprising administering an **antibody** to Death Domain Containing Receptor proteins and a second therapeutic agent.
DC B04 D16
IN DILLON, P J; DIXIT, V M; GENTZ, R L; NI, J; YU, G
PA (DILL-I) DILLON P J; (DIXI-I) DIXIT V M; (GENT-I) GENTZ R L; (HUMA-N) HUMAN GENOME SCI INC; (NIJJ-I) NI J; (UNMI) UNIV MICHIGAN; (YUGG-I) YU G
CYC 92
PI WO 2000064465 A1 20001102 (200067)* EN 265p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ
EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK
LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI
SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000044782 A 20001110 (200109)
ADT WO 2000064465 A1 WO 2000-US10741 20000421; AU 2000044782 A AU 2000-44782 20000421
FDT AU 2000044782 A Based on WO 200064465
PRAI US 1999-136741P 19990528; US 1999-130488P 19990422
AB WO 200064465 A UPAB: 20001223
NOVELTY - A method for treating graft-versus-host disease, cancer, immunodeficiency or an autoimmune disease comprising administering an

antibody to Death Domain Containing Receptor (DR3 and DR3-V1) proteins and a second therapeutic agent, is new.

DETAILED DESCRIPTION - A method for treating graft versus host disease, cancer, immunodeficiency or an autoimmune disease comprising administering an **antibody** to Death Domain Containing Receptor (DR3 and DR3-V1) proteins and a second therapeutic agent, is new. DR3 and DR3-V1 comprise defined 417 and 428 amino acid sequences, respectively and are members of the tumor necrosis factor (**TNF**) family of receptors. The second therapeutic agent is selected from a **TNF** blocking agent, an immunosuppressive agent, an antibiotic, an antiinflammatory agent, a chemotherapeutic agent and a cytokine.

An INDEPENDENT CLAIM is also included for an amino acid sequence (P1) comprising residues 36 to 212 of DR3-V1 covalently attached to polyethylene glycol (PEG) having a molecular weight of 2000 to 20000.

ACTIVITY - Immunosuppressive; cytostatic; cardiovascular general; cytostatic; antiarthritic; anti-diabetic; antiviral; neuroprotective; hepatotropic; vulnerary; osteopathic; antibacterial.

Experimental details are described but no results are given.

MECHANISM OF ACTION - Gene **therapy**.

Experimental details are described but no results are given.

USE - The method is useful for treating graft versus host disease, cancer, immunodeficiency or an autoimmune disease. P1 may be used to treat or diagnose a variety of conditions such as cardiovascular disorders, cancer, **arthritis**, diabetes, viral infections, neurodegenerative disease, liver disease, wounds, septic shock and osteoporosis.

Dwg.0/4

L18 ANSWER 274 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD

AN 2000-422868 [36] WPIDS

CR 1996-268530 [27]; 1998-377241 [29]; 2000-061893 [05]; 2000-071668 [05]; 2000-170770 [05]

DNC C2000-127890

TI Therapeutic **treatment** of for example viral diseases such as chronic hepatitis B and C, cancers such as leukemia, and multiple sclerosis comprises administering an immunological tolerance inducing compound prior to an effective drug .

DC B04 D16

IN TOVEY, M G

PA (PHAR-N) PHARMA PACIFIC PTY LTD

CYC 21

PI WO 2000032223 A2 20000608 (200036)* EN 26p

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE

W: AU JP US

AU 2000013991 A 20000619 (200044)

ADT WO 2000032223 A2 WO 1999-GB4009 19991201; AU 2000013991 A AU 2000-13991

19991201
FDT AU 2000013991 A Based on WO 200032223
PRAI EP 1998-403020 19981202
AB WO 200032223 A UPAB: 20000801
NOVELTY - Therapeutic **treatment** of a subject with an immunogenic drug comprising:
(a) administering oromucosally a first formulation comprising a compound which induces immunological tolerance to the drug; and
(b) administering a second formulation comprising the drug that effects the therapeutic **treatment**.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) A kit for therapeutic **treatment** of a subject with an immunogenic drug comprising a formulation comprising a compound to induce immunological tolerance to the drug and a formulation comprising the drug to effect the therapeutic **treatment**;
(2) Using an immunogenic drug for the manufacture of a formulation to effect therapeutic **treatment** of a disease of a human or animal which has become immunologically tolerant to the drug by the oromucosal route of a formulation comprising a compound that induces immunological tolerance; and
(3) Using a compound for the manufacture of a formulation for oromucosal administration to a human or animal to induce immunological tolerance to an immunological drug where the human or animal is also administered a second formulation comprising the drug to effect a therapeutic effect.
ACTIVITY - Virucide; Cytostatic; Neuroprotective; Immunostimulant; Antianemic; Antibacterial; Immunosuppressive; Antirheumatic; Antiarthritic.
MECHANISM OF ACTION - None given.
USE - For therapeutic **treatment** of a human or animal. An immunogenic drug or compound is used to manufacture formulations for inducing an immunological tolerance or effecting therapeutic **treatment** (claimed). Viral diseases, such as chronic hepatitis B and C, herpes, and influenza; cancers, such as leukemia, lymphomas and solid tumors; and multiple sclerosis are treated. Neutropenia and leukopenia following chemotherapy are treated. Anemia, chronic renal failure. septic shock and rheumatoid **arthritis** are treated. Cystic fibrosis and Gaucher disease can be treated by gene **therapy**.
ADVANTAGE - An immunological tolerance to an immunogenic drug is

induced so that when the drug is subsequently administered, its pharmacokinetics and/or clinical effectiveness are improved. Rejection of drugs that are administered in repeat doses over a period of time by the immune system is less likely. The amount of drug that needs to be administered is reduced, lowering costs. Non-humanized **antibodies** that cannot normally be used for **therapy** due to rejection by the immune system can be used.
Dwg.0/0

L18 ANSWER 275 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2000-182039 [16] WPIDS
DNN N2000-134380 DNC C2000-056809
TI A process for expanding and selecting disease associated T-cells useful for the production of vaccines.
DC B04 D16 S03
IN ANGHOLT, J; KALTOFT, K; AGNHOLT, J
PA (AGNH-I) AGNHOLT J; (KALT-I) KALTOFT K; (CELL-N) CELLCURE APS
CYC 87
PI WO 2000000587 A1 20000106 (200016)* EN 124p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB
GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU
LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR
TT UA UG US UZ VN YU ZA ZW
AU 9946034 A 20000117 (200026)
EP 1090104 A1 20010411 (200121) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
ADT WO 2000000587 A1 WO 1999-DK363 19990625; AU 9946034 A AU 1999-46034 19990625; EP 1090104 A1 EP 1999-929110 19990625, WO 1999-DK363 19990625
FDT AU 9946034 A Based on WO 200000587; EP 1090104 A1 Based on WO 200000587
PRAI US 1998-91684P 19980702; DK 1998-848 19980626; DK 1998-895 19980701
AB WO 200000587 A UPAB: 20000330
NOVELTY - A method (A) of expanding and selecting disease associated T-cells comprises: (a1) obtaining a tissue sample from a mammal including a human being, comprising disease activated T-cells, or (a2) obtaining T-cells and antigen-presenting cell from the mammal and mixing the cells with a disease associated antigen or antigens; and (b) culturing the tissue sample or the mixture of cells and antigen(s) in the presence of at

least 2 factors which promote T-cell growth and optionally at least 1 additional compound.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a vaccine comprising activated disease associated inflammatory T-cells prepared by (A);

(2) a pharmaceutical composition for use in an adjuvant **treatment** of a disease comprising disease associated regulatory or cytotoxic T-cells prepared by (A);

(3) a method for the diagnosis of a disease in a mammal, comprising:

(a) obtaining a tissue sample from a mammal including a human being, the sample comprising activated T-cells, antigen presenting cells and antigen(s); and

(b) culturing the tissue sample or the activated T-cells in the presence of two or more T-cell growth factors and optionally one or more additional compound; a method for the **treatment**, alleviation or prevention of a disease associated with an activation of T-cells in a subject comprising administering a T-cell line produced as described above;

(4) a model system for testing the effect of a medicament against a T-cell associated disease comprising at least one T-cell line as described above;

(5) a method for the **treatment**, alleviation or prevention of a disease associated with an activation of T-cells in a subject comprising administering (2); and

(6) a method of monitoring the response to a **treatment** of a disease of inflammatory, auto-immune, allergic, neoplastic or transplantation-related origin, or combinations thereof, comprising comparing the phenotype proliferation, apoptosis, cytokine profile, intracellular amount of NFKB and/or JAK/STAT pathway of activated Tcells in tissue sample taken from the patient to be treated before the start of the **treatment** and during the **treatment** and/or after the **treatment** has ended.

USE - The disease associated T-cells are associated with a disease of inflammatory, auto-immune, allergic, neoplastic and/or transplantation-related origin. The disease of inflammatory or allergic origin is a chronic inflammatory disease, or a chronic allergic disease.

The disease is an chronic inflammatory bowel disease, such as Crohn's

disease or ulcerative colitis, sclerosis, type I diabetes, rheumatoid
arthritis, psoriasis, atopic dermatitis, asthma, malignant melanoma, renal carcinoma, breast cancer, lung cancer, cancer of the uterus, prostatic cancer, cutaneous lymphoma, hepatic carcinoma, rejection-related disease, or Graft-versus-host-related disease. Dwg.0/22

L18 ANSWER 276 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 1998-272127 [24] WPIDS
CR 1996-105847 [11]; 2000-086224 [07]; 2001-217934 [18]; 2001-280761 [25]; 2001-380456 [38]
DNC C1998-084968
TI New immunostimulatory nucleic acid molecules - which contain at least one unmethylated CpG dinucleotide, used for treating e.g. tumours, infections or autoimmune disease.
DC B04 D16
IN KLINE, J N; KRIEG, A M; KLINMAN, D; STEINBERG, A; WEINER, G
PA (IOWA) UNIV IOWA RES FOUND
CYC 79
PI WO 9818810 A1 19980507 (199824)* EN 109p
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9852424 A 19980522 (199840)
EP 948510 A1 19991013 (199947) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
CN 1235609 A 19991117 (200013)
NZ 335397 A 20001124 (200065)
JP 2001503267 W 20010313 (200117) 110p
KR 2000052994 A 20000825 (200121)
ADT WO 9818810 A1 WO 1997-US19791 19971030; AU 9852424 A AU 1998-52424 19971030; EP 948510 A1 EP 1997-947311 19971030, WO 1997-US19791 19971030; CN 1235609 A CN 1997-199352 19971030; NZ 335397 A NZ 1997-335397 19971030, WO 1997-US19791 19971030; JP 2001503267 W WO 1997-US19791 19971030, JP 1998-520784 19971030; KR 2000052994 A WO 1997-US19791 19971030, KR 1999-703873 19990430
FDT AU 9852424 A Based on WO 9818810; EP 948510 A1 Based on WO 9818810; NZ 335397 A Based on WO 9818810; JP 2001503267 W Based on WO 9818810; KR 2000052994 A Based on WO 9818810

PRAI US 1996-738652 19961030
AB WO 9818810 A UPAB: 20010719
An isolated nucleic acid (NA) sequence (A) which contains at least one
unmethylated CpG dinucleotide, having formula (I):
5' N1X1CGX2N2 3' (I);
where at least one nucleotide separates consecutive CpGs; X1 is
adenine, guanine, or thymine; X2 is cytosine or thymine; N is any
nucleotide and N1 + N2 is 0-26 bases with the provisoin that N1 and N2
does not contain a CCGG tetramer or more than one CCG or CGG trimer; and
the NA sequence is 8-30 bases in length.
Also claimed are: (1) an isolated NA sequence containing at least one
unmethylated CpG dinucleotide and having formula (II):
5' NX1X2CGX3X4N 3' (II);
where at least one nucleotide separates consecutive CpGs; X1 and X2
are selected from GpT, GpG, GpA, ApT and ApA; X3and X4 are selected from
TpT or CpT; N is any nucleotide and N1 + N2 is 0-26 bases with the
provision that N1 and N2 does not contain a CCGG tetramer or more than one
CCG or CGG trimer; and the NA sequence is 8-30 bases in length; and (2) a
method for treating a subject having an autoimmune or other CpG associated
disorder by inhibiting CpG-mediated leukocyte activation, comprising
administering to the subject an inhibitor of endosomal acidification, in a
carrier.
USE - The nucleic acids activate lymphocytes in a subject and
redirect a subject's immune response from a Th2 to a Th1 (e.g. by inducing
monocytic cells and other cells to produce Th1 cytokines, including
IL-12, IFN-gamma and GM-CSF). By redirecting a
subject's immune response from TH2 to Th1, products can be used to treat
or prevent an asthmatic disorder. In addition, the products can be
administered to a subject in conjunction with a particular allergen as a
type of desensitisation **therapy** to treat or prevent the
occurrence of an allergic reaction associated with an asthmatic disorder.
They can be used as an artificial adjuvant during **antibody**
generation in a mammal such as a mouse or a human. They can also be used
to treat immune system deficiencies. They can be used to treat disorders
such as tumours or a viral, fungal, bacterial or parasitic infection. The
NA (A) or as described in (1) can be used to stimulate cytokine production

especially **IL-12**, IL-6, IFN-g, **TNF**- alpha ,
and GM-CSF or may be used to stimulate NK lytic activity or B cell
proliferation in humans(all claimed). (A) or the NA as in (1) may also be
used to treat asthamatic disorder or may be used as an adjuvant (all
claimed). Autoimmune diseases or other CpG associatted disorders can be
treated by inhibbitting CpG mediatted leukocyte activation using
inhibitors of endosomal acidification e.g. to treat disorders such as
systemic lupus erythematosus, sepsis, inflammatory bowel disease,
psoriasis, gingivitis, **arthritis**, Crohn's disease, Grave's
disease or asthma (all claimed).
Dwg.0/15

L18 ANSWER 277 OF 281 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 1998-261495 [23] WPIDS
DNC C1998-081292
TI New compositions for immuno-**therapy** and protection - comprise
nucleotide sequences encoding an immuno-modulating protein and an antigen,
used for e.g. infections, cancer or auto-immune diseases.
DC B04 C06 D16
IN BAGARAZZI, M L; BOYER, J D; KIM, J J; WANG, B; WEINER, D B; AYYAVOO, V
PA (APOL-N) APOLLON INC; (UYPE-N) UNIV PENNSYLVANIA
CYC 80
PI WO 9817799 A1 19980430 (199823)* EN 136p
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT
SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE
GH HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN
MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ
VN YU ZW
AU 9750022 A 19980515 (199838)
EP 958364 A1 19991124 (199954) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
BR 9712852 A 19991116 (200012)
CN 1242045 A 20000119 (200023)
AU 729579 B 20010201 (200112)
KR 2000052710 A 20000825 (200121)
JP 2001507216 W 20010605 (200138) 141p
ADT WO 9817799 A1 WO 1997-US19502 19971023; AU 9750022 A AU 1997-50022
19971023; EP 958364 A1 EP 1997-912961 19971023, WO 1997-US19502 19971023;
BR 9712852 A BR 1997-12852 19971023, WO 1997-US19502 19971023; CN 1242045
A CN 1997-180897 19971023; AU 729579 B AU 1997-50022 19971023; KR 2000052710 A WO 1997-US19502 19971023, KR 1999-703507 19990422; JP
2001507216 W WO 1997-US19502 19971023, JP 1998-519714 19971023

FDT AU 9750022 A Based on WO 9817799; EP 958364 A1 Based on WO 9817799; BR
9712852 A Based on WO 9817799; AU 729579 B Previous Publ. AU 9750022,
Based on WO 9817799; KR 2000052710 A Based on WO 9817799; JP 2001507216 W
Based on WO 9817799
PRAI US 1996-28613P 19961023
AB WO 9817799 A UPAB: 19980610
The following are claimed: (A) A plasmid which comprises a nucleotide
sequence (NS) that encodes:(a) an immunomodulating protein selected from
interleukin (**IL**)-**12**, granulocyte-macrophage colony
stimulating factor (GM-CSF), IL-1, tumour necrosis factor (**TNF**)-
alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and
BL-1 operably linked to regulatory elements; (b) a NS that encodes an
immunogen; (B) a composition comprising at least 2 plasmids including a
first plasmid comprising a NS that encoded an immunomodulating protein
selected from **IL-12**, GM-CSF, IL-1, **TNF**-
alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and
BL-1 operably linked to regulatory elements; and a second plasmid
comprising a NS that encodes an immunogen; (C) a recombinant vaccine
comprising a NS that encodes an immunomodulating protein selected from
IL-12, GM-CSF, IL-1, **TNF**- alpha , **TNF**
- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and BL-1 operably linked to
regulatory elements; and a second plasmid comprising a NS that encodes an
immunogen; (D) a live attenuated pathogen comprising a NS that encodes an
immunomodulating protein selected from **IL-12**, GM-CSF,
IL-1, **TNF**- alpha , **TNF**- beta , IL-2, IL-4, IL-5,
IL-10, IL-15, IL-18, and BL-1 operably linked to regulatory elements; (E)
a plasmid comprising a NS that encodes single chain **IL-12**; (F) a pure BL-1 protein having an amino acid sequence given in
the specification, or an immunomodulatory fragment; (G) a recombinant
expression vector comprising a nucleic acid sequence that encodes a
protein as in (F); (H) an isolated **antibody** which binds to an
epitope on a protein as in (F).

The immunogen in (A) is a target protein operably linked to
regulatory segments, where the target protein encodes a pathogen antigen,
a cancer-associated antigen or an antigen linked to cells associated with
autoimmune diseases. It is preferably an HIV-1 antigen. The
immunomodulatory protein is a single chain **IL-12**. The
antibody (H) is a monoclonal **antibody**.

USE - The products can be used to induce an immune response to an

antigen such as a pathogen antigen, a hyperproliferative disease-associated antigen, and antigen linked to cells associated with autoimmune diseases or an allergen. They can be used for immunotherapy or to provide a protective immune response. In particular, they can be used for treating subjects with an allergic reaction, pathogen infection, hyperproliferative disease such as cancer or psoriasis or autoimmune diseases e.g. rheumatoid **arthritis** (RA), multiple sclerosis (MS), Sjogren's syndrome, sarcoidosis, insulin dependent diabetes mellitus (IDDM), autoimmune thyroiditis, reactive **arthritis**, ankylosing spondylitis, scleroderma, polymyositis, dermatomyositis, psoriasis, vasculitis, Wegener's granulomatosis, Crohn's disease and ulcerative colitis, lupus (SLE), Grave's disease, myasthenia gravis, autoimmune haemolytic anaemia, autoimmune thrombocytopenia, asthma, cryoglobulinaemia, primary biliary sclerosis and pernicious anaemia.
Dwg.0/17

L18 ANSWER 278 OF 281 CAPLUS COPYRIGHT 2002 ACS
AN 2000:688272 CAPLUS
DN 133:280563
TI Human **antibodies** that bind human **IL-12** and methods for producing
IN Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PA Basf A.-G., Germany; Genetics Institute Inc.; et al.
SO PCT Int. Appl., 377 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000056772	A1	20000928	WO 2000-US7946	20000324

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA,

ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRAI US 1999-126603 P 19990325
AB Human **antibodies**, preferably recombinant human **antibodies**, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred **antibodies** have high affinity for hIL-12 and neutralize hIL-12 activity in vitro and in vivo . An **antibody** of the invention can be a full-length **antibody** or an antigen-binding portion thereof. The **antibodies**, or **antibody** portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human **antibodies** of the invention, and methods of synthesizing the recombinant human **antibodies**, are also encompassed by the invention.
RE.CNT 7
RE
(2) Carter, R; HYBRIDOMA 1997, V16(4), P363 CAPLUS
(3) Genentech Inc; WO 9404679 A 1994 CAPLUS
(4) Genetics Inst; WO 9524918 A 1995 CAPLUS
(5) Irving, R; IMMUNOTECHNOLOGY 1996, V2(2), P127 CAPLUS
(6) Pini, A; JOURNAL OF IMMUNOLOGICAL METHODS 1997, V206(1-2), P171 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 279 OF 281 CAPLUS COPYRIGHT 2002 ACS
AN 1995:934127 CAPLUS
DN 123:337469
TI Use of **IL-12** and **IL-12** **antagonists** in **treatment** of autoimmune diseases
IN Leonard, John P.; Goldman, Samuel; O'Hara, Richard, Jr.
PA Genetics Institute, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

	JP 09510444	T2	19971021	JP 1995-524044	19950307
	US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		

AB Autoimmune conditions such as multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or **TNF**-.alpha., are treated in mammals by administering **IL-12** or an **IL-12 antagonist**. Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with **IL-12** during restimulation, and alleviated by injection of a polyclonal **antibody** to **IL -12**.

L18 ANSWER 280 OF 281 LIFESCI COPYRIGHT 2002 CSA
AN 2000:98861 LIFESCI
TI Gene **therapy** of autoimmune diseases with vectors encoding regulatory cytokines or inflammatory cytokine inhibitors
AU Prud'homme, G.J.
CS Department of Pathology, McGill University, 3775 University St, Rm B13, Montreal, Quebec, H3A 2B4, Canada; E-mail: gprudh@po-box.mcgill.ca
SO Journal of Gene Medicine [J. Gene Med.], (20000800) vol. 2, no. 4, pp. 222-232.
ISSN: 1099-498X.
DT Journal
TC General Review
FS W3; G
LA English
SL English
AB Gene **therapy** offers advantages for the immunotherapeutic delivery of cytokines or their inhibitors. After gene transfer, these mediators are produced at relatively constant, non-toxic levels and sometimes in a tissue-specific manner, obviating limitations of protein administration. **Therapy** with viral or nonviral vectors is effective in several animal models of autoimmunity including Type 1 diabetes mellitus (DM), experimental allergic encephalomyelitis (EAE), systemic lupus erythematosus (SLE), colitis, thyroiditis and various forms

of **arthritis**. Genes encoding transforming growth factor beta , interleukin-4 (IL-4) and IL-10 are most frequently protective. Autoimmune/inflammatory diseases are associated with excessive production of inflammatory cytokines such as IL-I, **IL-12**, tumor necrosis factor alpha (**TNF** alpha) and interferon gamma (IFN gamma). Vectors encoding inhibitors of these cytokines, such as IL-1 receptor **antagonist**, soluble IL-1 receptors, IL-12p40, soluble **TNF** alpha receptors or IFN gamma -receptor/IgG-Fc fusion proteins are protective in models of either **arthritis**, Type 1 DM, SLE or EAE. We use intramuscular injection of naked plasmid DNA for cytokine or anticytokine **therapy**. Muscle tissue is accessible, expression is usually more persistent than elsewhere, transfection efficiency can be increased by low-voltage in vivo electroporation, vector administration is simple and the method is inexpensive. Plasmids do not induce neutralizing immunity allowing repeated administration, and are suitable for the **treatment** of chronic immunological diseases.

L18 ANSWER 281 OF 281 BIOTECHDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-08257 BIOTECHDS
TI Composition containing interleukin-12 p40 and IL-B30 protein or its segment, useful for ameliorating rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;
vector-mediated gene transfer and expression in host cell, **antibody** and **antagonist**
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K
PA Schering-USA
LO Kenilworth, NJ, USA.
PI WO 2001018051 15 Mar 2001
AI WO 2000-US24686 8 Sep 2000
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999
DT Patent
LA English
OS WPI: 2001-244560 [25]
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous amino acids from **IL-12** p40 and/or IL-B30, is new. Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural

mature coding portion of primate **IL-12** p40 and
IL-B30; an **antagonist** of **IL-12** p40/IL-B30
combined with a tumor necrosis factor-alpha (**TNF**-alpha)
antagonist, an **IL-12 antagonist**,
IL-10 or steroids; a binding compound containing an antigen binding site
from an **antibody** which specifically binds to the protein; a kit
containing the composition, polynucleotide and a binding compound;
producing an antigen:**antibody** complex; a composition containing
a binding compound; increasing the secretion of a primate IL-B30; and
screening for a receptor which binds the composition. The composition is
useful for modulating physiology or development of a cell or tissue0.
(69pp)

=> d his

(FILE 'HOME' ENTERED AT 12:31:11 ON 18 JAN 2002)

FILE 'EMBASE, MEDLINE, BIOSIS, USPATFULL, JAPIO, WPIDS, CAPLUS, AGRICOLA,
LIFESCI, BIOTECHDS, JICST-EPLUS' ENTERED AT 12:31:20 ON 18 JAN 2002

		E	LEONARD JOHN P/AU
L1	90	S	E3-E5
		E	LEONARD J P/AU
L2	350	S	E3-E4
		E	GOLDMAN SAMUEL/AU
L3	79	S	E1-E9
		E	GOLDMAN S/AU
L4	1413	S	E3
		E	OHARA RICHARD/AU
		E	O HARA RICHARD/AU
L5	25	S	E3-E7
		E	O HARA R/AU
L6	78	S	E3
L7	48	S	E11
L8	2069	S	L1-L7
L9	23	S	L8 AND ARTHRITIS
L10	4	S	L9 AND IL-12
L11	12	DUP REM	L9 (11 DUPLICATES REMOVED)
L12	60	S	L8 AND (IL-12 OR NKSF OR CLMF)
L13	24	DUP REM	L12 (36 DUPLICATES REMOVED)
L14	1030	S	ARTHRITIS AND (IL-12 OR NKSF OR CLMF)
L15	492	S	L14 AND TNF
L16	345	S	L15 AND (ANTAGONIST? OR ANTIBOD?)
L17	301	DUP REM	L16 (44 DUPLICATES REMOVED)
L18	281	S	L17 AND (THERAPY OR TREATMENT)

=> s l18 and sclerosis

L19 178 L18 AND SCLEROSIS

=> d bib ab 1-178

L19 ANSWER 1 OF 178 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 93264853 EMBASE
DN 1993264853
TI Clinical and preclinical studies presented at the keystone symposium on
arthritis, related diseases, and cytokines.
AU Ralph P.
CS Department of Immunology, Genentech, Inc., 460 Point San Bruno Avenue,South San Francisco, CA 94080, United States
SO Lymphokine and Cytokine Research, (1993) 12/4 (261-263).
ISSN: 0277-6766 CODEN: LCREEY
CY United States
DT Journal; Conference Article
FS 006 Internal Medicine
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Topics include **treatment** of multiple **sclerosis** (MS) with T cell receptor (TCR) peptides, rheumatoid **arthritis** (RA) with IL-1ra, IL-2 toxin conjugate, or **antibodies** to **TNF**, to CD4, or to ICAM-1, sepsis and five other diseases with IL-1ra, and
treatment of experimental animal diseases with soluble receptors, **IL-12**, TGF-.beta.2, or small molecule **antagonists** of cytokines.

L19 ANSWER 2 OF 178 USPATFULL
AN 2002:9923 USPATFULL
TI Interleukin-1 Hy2 materials and methods
IN Ballinger, Dennis G., Menlo Park, CA, United States
Pace, Ann M., Scots Valley, CA, United States
PA Hycey Inc., Sunnydale, CA, United States (U.S. corporation)
PI US 6339141 B1 20020115
AI US 1999-316081 19990520 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Stucker, Jeffrey; Assistant Examiner: Seharaseyon, Jegatheesan
LREP Marshall, Gerstein, & Borun
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 4019
AB The present invention provides novel nucleic acids encoding IL-1 Hy2, a novel member of the Interleukin-1 Receptor **Antagonist** family, the novel polypeptides encoded by these nucleic acids and uses of these and related products.

L19 ANSWER 3 OF 178 USPATFULL
AN 2002:9647 USPATFULL
TI Use of **IL-12** and **IL-12** **antagonists** in the **treatment** of autoimmune diseases

IN Leonard, John, Auburn, NH, United States
Goldman, Samuel, Acton, MA, United States
O'Hara, Jr., Richard, Quincy, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6338848 B1 20020115
AI US 2000-513380 20000225 (9)
RLI Continuation of Ser. No. US 1995-560943, filed on 20 Nov 1995, now abandoned Continuation of Ser. No. US 1994-212629, filed on 14 Mar 1994, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Minnifield, Nita M.
LREP Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 676
AB Method of treating autoimmune conditions are disclosed comprising administering to a mammalian subject **IL-12** or an **IL-12 antagonist**. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels of IFN-.gamma. or **TNF**-.alpha.. Suitable conditions for **treatment** include multiple **sclerosis**, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and autoimmune inflammatory eye disease.

L19 ANSWER 4 OF 178 USPATFULL
AN 2002:8587 USPATFULL
TI Multivalent **antibodies** and uses therefor
IN Miller, Kathy L., San Francisco, CA, UNITED STATES
Presta, Leonard G., San Francisco, CA, UNITED STATES
PA GENENTECH, INC. (U.S. corporation)
PI US 2002004587 A1 20020110
AI US 2001-813341 A1 20010320 (9)
PRAI US 2000-195819 20000411 (60)
DT Utility
FS APPLICATION
LREP Attn: Wendy M. Lee, 1 DNA Way, South San Francisco, CA, 94080-4990
CLMN Number of Claims: 93
ECL Exemplary Claim: 1
DRWN 45 Drawing Page(s)
LN.CNT 4913
AB The present application describes engineered **antibodies**, with three or more functional antigen binding sites, and uses, such as therapeutic applications, for such engineered **antibodies**.

L19 ANSWER 5 OF 178 USPATFULL
AN 2002:8489 USPATFULL

TI Retinoid receptor interacting polynucleotides, polypeptides, and
antibodies
IN Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002004489 A1 20020110
AI US 2001-788600 A1 20010221 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US22351, filed on 15 Aug 2000,
UNKNOWN
PRAI US 1999-148757 19990816 (60)
US 2000-189026 20000314 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 11257
AB The present invention relates to novel human RIP polypeptides and
isolated nucleic acids containing the coding regions of the genes
encoding such polypeptides. Also provided are vectors, host cells,
antibodies, and recombinant methods for producing human RIP
polypeptides. The invention further relates to diagnostic and
therapeutic methods useful for diagnosing and treating disorders related
to these novel human RIP polypeptides.

L19 ANSWER 6 OF 178 USPATFULL
AN 2002:8044 USPATFULL
TI Methods for abrogating a cellular immune response
IN Albert, Matthew L., New York, NY, UNITED STATES
Jegathesan, Mithila, New York, NY, UNITED STATES
Darnell, Robert B., Pelham, NY, UNITED STATES
PI US 2002004041 A1 20020110
AI US 2001-804584 A1 20010312 (9)
RLI Continuation-in-part of Ser. No. US 2000-545958, filed on 10 Apr 2000,
PENDING Continuation-in-part of Ser. No. US 1999-251896, filed on 19 Feb
1999, PENDING
DT Utility
FS APPLICATION
LREP KLAUBER & JACKSON, 411 HACKENSACK AVENUE, HACKENSACK, NJ, 07601
CLMN Number of Claims: 41
ECL Exemplary Claim: 1
DRWN 19 Drawing Page(s)
LN.CNT 1598
AB Methods are provided for preventing a cellular immune response to a
pre-selected antigen by ex vivo or in vivo methods whereby dendritic
cell maturation is permitted to occur in the absence of effective CD4+ T
cell help. Under these conditions, elimination of cytotoxic T cells is

achieved. The methods may be used for the prophylaxis of an undesired
immune response to an autoimmune disease antigen, a transplant antigen,
or reducing an exaggerated immune response to a antigen.

L19 ANSWER 7 OF 178 USPATFULL
AN 2002:5759 USPATFULL
TI Interleukin-1 receptor **antagonist** and recombinant production thereof
IN Ford, John, San Mateo, CA, United States
Pace, Ann, Scotts Valley, CA, United States
PA Hyseq, Inc., Sunnyvale, CA, United States (U.S. corporation)
PI US 6337072 B1 20020108
AI US 1999-348942 19990707 (9)
RLI Continuation-in-part of Ser. No. US 1999-287210, filed on 5 Apr 1999, now abandoned Continuation-in-part of Ser. No. US 1999-251370, filed on 17 Feb 1999, now abandoned Continuation-in-part of Ser. No. US 1999-229591, filed on 13 Jan 1999, now abandoned Continuation-in-part of Ser. No. US 1998-127698, filed on 31 Jul 1998, now abandoned Continuation of Ser. No. US 1998-99818, filed on 19 Jun 1998, now abandoned Continuation of Ser. No. US 1998-82364, filed on 20 May 1998, now abandoned Continuation-in-part of Ser. No. US 1998-79909, filed on 15 May 1998, now abandoned Continuation-in-part of Ser. No. US 1998-55010, filed on 3 Apr 1998, now abandoned
PRAI WO 1999-US4291 19990405
DT Utility
FS GRANTED
EXNAM Primary Examiner: Spector, Lorraine
LREP Marshall, O'Toole, Gerstein, Murray & Borun
CLMN Number of Claims: 37
ECL Exemplary Claim: 1,15
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 5025
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel nucleic acids, the novel polypeptide sequences encoded by these nucleic acids and uses thereof.
These novel polynucleotide and polypeptide sequences were determined to
be a novel Interleukin-1 Receptor **Antagonist**.

L19 ANSWER 8 OF 178 USPATFULL
AN 2002:3866 USPATFULL
TI CONTINUOUS T-CELL LINES
IN KALTOFT, KELD, HAMMEL, DENMARK
AGNHOLT, JORGEN, RISSKOV, DENMARK
PI US 2002001841 A1 20020103
AI US 1999-339836 A1 19990625 (9)
PRAI DK 1998-848 19980626
DK 1998-895 19980701
US 1998-91684 19980702 (60)
DT Utility

FS APPLICATION
LREP JACOBSON PRICE HOLMAN & STERN, 400 SEVENTH STREET NW, WASHINGTON, DC, 20004
CLMN Number of Claims: 84
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 2563
AB Methods of expanding and selecting disease associated T-cells, continuous T-cell lines as well as T-cell lines obtainable by these methods are disclosed. Furthermore, pharmaceutical compositions and vaccines comprising activated disease associated T-cell are disclosed.
The uses of the T-cells and T-cell lines are numerous and include methods of diagnosis, methods for the **treatment**, alleviation or prevention of diseases associated with activation of T-cells, methods of testing the effect of medicaments against T-cell associated diseases, methods of detecting T-cell growth factors, methods of monitoring the response to **treatment**, alleviation or prevention of diseases associated with activation of T-cells, and methods of identifying disease associated antigens.

L19 ANSWER 9 OF 178 USPATFULL
AN 2002:1314 USPATFULL
TI T-cell selective interleukin-4 agonists
IN Shanafelt, Armen B., Moraga, CA, United States
Greve, Jeffrey M., Berkeley, CA, United States
Gundel, Robert, Alamo, CA, United States
PA Bayer Corporation, Berkeley, CA, United States (U.S. corporation)
PI US 6335426 B1 20020101
AI US 1999-298374 19990423 (9)
RLI Continuation-in-part of Ser. No. US 1997-874697, filed on 13 Jun 1997, now patented, Pat. No. US 5986059
PRAI US 1997-36746 19970127 (60)
US 1996-19748 19960614 (60)
US 1997-36746 19970127 (60)
US 1996-19748 19960614 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Kunz, Gary L.; Assistant Examiner: Landsman, Robert S.
LREP Mahoney, John W., Shaw, Melissa A.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 33 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 2191
AB This invention realizes a less toxic IL-4 mutant that allows greater therapeutic use of interleukin 4. Further, the invention is directed to

IL-4 muteins having single and double mutations represented by the designators R121E and T13D/R121E, numbered in accordance with wild type IL-4 (His=1). The invention also includes polynucleotides coding for the muteins of the invention, vectors containing the polynucleotides, transformed host cells, pharmaceutical compositions comprising the muteins, and therapeutic methods of **treatment**.

L19 ANSWER 10 OF 178 USPATFULL
AN 2002:926 USPATFULL
TI Methods and materials relating to CD39-like polypeptides
IN Ford, John, San Mateo, CA, United States
Mulero, Julio J., Palo Alto, CA, United States
Yeung, George, Mountain View, CA, United States
PA Hyseq, Inc., Sunnyvale, CA, United States (U.S. corporation)
PI US 6335013 B1 20020101
AI US 2000-608285 20000630 (9)
RLI Continuation-in-part of Ser. No. US 2000-583231, filed on 26 May 2000
Continuation-in-part of Ser. No. US 2000-557800, filed on 25 Apr 2000
Continuation-in-part of Ser. No. US 2000-481238, filed on 11 Jan 2000
Continuation-in-part of Ser. No. US 1999-370265, filed on 9 Aug 1999
Continuation-in-part of Ser. No. WO 1999-US16180, filed on 16 Jul 1999
Continuation-in-part of Ser. No. US 1999-350836, filed on 9 Jul 1999
Continuation-in-part of Ser. No. US 1999-273447, filed on 19 Mar 1999
DT Utility
FS GRANTED
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: DeCloux, Amy
LREP Marshall, Gerstein & Borun
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 11 Drawing Page(s)
LN.CNT 4738
AB The invention provides novel polynucleotides isolated from cDNA libraries of human fetal liver-spleen and macrophage as well as polypeptides encoded by these polynucleotides and mutants or variants thereof. The polypeptides correspond to a novel human CD39-like protein.
Other aspects of the invention include vectors containing polynucleotides of the invention and related host cells as well a processes for producing novel CD39-like polypeptides, and **antibodies** specific for such polypeptides.

L19 ANSWER 11 OF 178 USPATFULL
AN 2001:235250 USPATFULL
TI Method of treating cytokine mediated diseases or conditions

IN Cirillo, Pier F., Woodbury, CT, United States
Gilmore, Thomas A., Middlebury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Regan, John R., Larchmont, NY, United States
Zhang, Lin-Hua, New Fairfield, CT, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United
States (U.S. corporation)
PI US 6333325 B1 20011225
AI US 2001-871559 20010531 (9)
RLI Continuation of Ser. No. US 2000-484638, filed on 18 Jan 2000
PRAI US 1999-116400 19990119 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Ramsuer, Robert W.
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2234
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel aromatic heterocyclic compounds of the formula(I)
wherein Ar.sub.1,Ar.sub.2,L,Q and X are described herein. The compounds
are useful in pharmaceutic compositions for treating diseases or
pathological conditions involving inflammation such as chronic
inflammatory diseases. Also disclosed are processes of making such
compounds. ##STR1##

L19 ANSWER 12 OF 178 USPATFULL
AN 2001:233534 USPATFULL
TI Method and composition for modulating an immune response
IN Salzman, Andrew, Belmont, MA, United States
Szabo, Csaba, Gloucester, MA, United States
PI US 2001053763 A1 20011220
AI US 2001-817829 A1 20010326 (9)
RLI Continuation-in-part of Ser. No. US 2000-626602, filed on 27 Jul 2000,
PENDING Continuation-in-part of Ser. No. US 2000-491888, filed on 24 Jan
2000, PENDING Continuation-in-part of Ser. No. US 1999-452427, filed on
1 Dec 1999, PENDING
PRAI US 1998-110562 19981202 (60)
DT Utility
FS APPLICATION
LREP MINTZ, LEVIN, COHN, FERRIS, GLOVSKY AND POPEO, P.C., One Financial
Center, Boston, MA, 02111
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 5 Drawing Page(s)
LN.CNT 1038
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a method of inhibiting or preventing a condition associated

with undesired secretion of a macrophage inflammatory protein using
inhibitors of ATP-sensitive K.sup.+-channels, inhibitors of the Na.sup.+/H.sup.+ antiporter, inosine, or inosine analogs.

L19 ANSWER 13 OF 178 USPATFULL
AN 2001:233136 USPATFULL
TI Novel amphipathic aldehydes and their uses as adjuvants and immunoeffectors
IN Johnson, David A., Hamilton, MT, United States
PI US 2001053363 A1 20011220
AI US 2001-810915 A1 20010316 (9)
PRAI US 2000-190466 20000317 (60)
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, TWO EMBARCADERO CENTER, EIGHTH FLOOR,
SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 47
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to novel aldehyde containing compounds and their
uses as adjuvants and immunoeffectors.

L19 ANSWER 14 OF 178 USPATFULL
AN 2001:226669 USPATFULL
TI Aromatic heterocyclic compounds as antiinflammatory agents
IN Cirillo, Pier F., Woodbury, CT, United States
Regan, John R., Larchmont, NY, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United
States (U.S. corporation)
PI US 6329415 B1 20011211
AI US 2001-891579 20010626 (9)
RLI Division of Ser. No. US 2000-484638, filed on 18 Jan 2000
PRAI US 1999-116400 19990101 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Ramsuer, Robert W.
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2204
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel aromatic heterocyclic compounds of the formula(I)
wherein Ar.sub.1,Ar.sub.2,L,Q and X are described herein. The compounds
are useful in pharmaceutic compositions for treating diseases or
pathological conditions involving inflammation such as chronic inflammatory diseases. Also disclosed are processes of making such
compounds. ##STR1##

L19 ANSWER 15 OF 178 USPATFULL
AN 2001:226622 USPATFULL
TI Inhibitors of interleukin-1.beta. converting enzyme
IN Golec, Julian M. C., Swindon, United Kingdom
Lauffer, David J., Stow, MA, United States
Livingston, David J., Lawrenceville, NJ, United States
Mullican, Michael D., Needham, MA, United States
Murcko, Mark A., Holliston, MA, United States
Nyce, Philip L., Millbury, MA, United States
Robidoux, Andrea L. C., Andover, MA, United States
Wannamaker, Marion W., Stow, MA, United States
PA Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6329365 B1 20011211
AI US 1999-326495 19990604 (9)
RLI Continuation of Ser. No. WO 1997-US22289, filed on 5 Dec 1997
PRAI US 1997-53001 19970626 (60)
US 1997-42660 19970404 (60)
US 1996-32792 19961206 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Kifle, Bruck
LREP Fish & Neave, Haley, Jr., James F., Joslyn, Kristin M.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2114
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel classes of compounds which are inhibitors of interleukin-1.beta. converting enzyme ("ICE"). This invention also relates to pharmaceutical compositions comprising these compounds. The compounds and pharmaceutical compositions of this invention are particularly well suited for inhibiting ICE activity and consequently, may be advantageously used as agents against interleukin-1-("IL-1"), apoptosis-, interferon-.gamma. inducing factor-(IGIF), interferon-.gamma.-("IFN-.gamma.") mediated diseases, excess dietary alcohol intake diseases, or viral diseases, including inflammatory diseases, autoimmune diseases, destructive bone disorders, proliferative disorders, infectious diseases, and degenerative diseases.
This invention also relates to methods for inhibiting ICE activity and decreasing IGIF production and IFN-.gamma. production and methods for treating interleukin-1, apoptosis- and interferon-.gamma.-mediated diseases using the compounds and compositions of this invention. This invention also relates to methods of preparing the compounds of this

invention.

L19 ANSWER 16 OF 178 USPATFULL
AN 2001:226258 USPATFULL
TI Methods for the **treatment** of immunologically-mediated skin disorders
IN Watson, James D., Auckland, New Zealand
Tan, Paul L. J., Auckland, New Zealand
Prestidge, Ross, Auckland, New Zealand
PA Genesis Research & Development Corp. Ltd., Parnell, New Zealand (non-U.S. corporation)
PI US 6328978 B1 20011211
AI US 1999-324542 19990602 (9)
RLI Continuation-in-part of Ser. No. US 1997-997080, filed on 23 Dec 1997,
now patented, Pat. No. US 5968524
DT Utility
FS GRANTED
EXNAM Primary Examiner: Devi, S.
LREP Sleath, Janet, Speckman, Ann W.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2453
AB Methods for the **treatment** of skin disorders, including psoriasis, atopic dermatitis, allergic contact dermatitis, alopecia areata and skin cancers are provided, such methods comprising administering a composition having antigenic and/or adjuvant properties.
Compositions which may be usefully employed in the inventive methods include inactivated M. vaccae cells, delipidated and deglycolipidated M. vaccae cells, M. vaccae culture filtrate and compounds present in or derived therefrom, together with combinations of such compositions.

L19 ANSWER 17 OF 178 USPATFULL
AN 2001:221075 USPATFULL
TI Retinoid **antagonists** and use thereof
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Mohr, Peter, Basel, Switzerland
Panina-Bordignon, Paola, Milan, Italy
Rosenberger, Michael, Caldwell, NJ, United States
Sinigaglia, Francesco, Milan, Italy
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6326397 B1 20011204
AI US 1999-307009 19990507 (9)
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998
DT Utility
FS GRANTED
EXNAM Primary Examiner: Killos, Paul J.
LREP Johnston, George W., Parise, John P.
CLMN Number of Claims: 16

ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1573
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel retinoid **antagonists** of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.sup.1 is lower alkyl and R.sup.2 is hydrogen; and, when the dotted bond is hydrogenated, R.sup.1 and R.sup.2 taken together are methylene to form a cis-substituted cyclopropyl ring; R.sup.3 is hydroxy or lower alkoxy; R.sup.4 is alkyl or alkoxy; and R.sup.5 and R.sup.6 are, independently, a C.sub.4-12 alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.sup.5 and R.sup.6 being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocylic acids of the formula I; as well as method for the **treatment** of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist **treatment** by administering a retinoid **antagonist**.

L19 ANSWER 18 OF 178 USPATFULL
AN 2001:218177 USPATFULL
TI Method of identifying the function of a test agent
IN Powell, Thomas J., Madison, CT, United States
Minskoff, Stacey A., Stamford, CT, United States
Quinn, Kerry E., Hamden, CT, United States
Ramesh, Tennore M., New Milford, CT, United States
PI US 2001046665 A1 20011129
AI US 2001-766863 A1 20010119 (9)
PRAI US 2000-177416 20000121 (60)
DT Utility
FS APPLICATION
LREP Ivor R. Elrifi, Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 406
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a method of identifying the function of a test compound by contacting a plurality of cells with the test compound. The plurality

includes at least a first cell and a second cell of a different type
than the first cell type. Expression of one or more genes in cells of
the plurality is measured. An alteration in the expression of the genes
relative to the expression of said one or more genes in a reference cell
reveals the function of said test compound

L19 ANSWER 19 OF 178 USPATFULL
AN 2001:215173 USPATFULL
TI Nucleic acid molecules encoding a 103 gene product and uses therefor
IN Kingsbury, Gillian A., W Roxbury, MA, United States
Leiby, Kevin R., Natick, MA, United States
PA Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S.
corporation)
PI US 6323334 B1 20011127
AI US 2000-560639 20000428 (9)
PRAI US 1999-155862 19990924 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Gambel, Phillip; Assistant Examiner: Roark, Jessica A.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 67 Drawing Figure(s); 33 Drawing Page(s)
LN.CNT 6541
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods and compositions for the
treatment and diagnosis of immune disorders, especially T helper
lymphocyte-related disorders. In particular, the invention provides a
nucleotide sequence which encodes a previously unknown human 103 gene
product. The invention also provides expression vectors containing the
nucleic acid molecules of the invention and host cells into which the
expression vectors have been introduced. The invention still further
provides isolated polypeptides, fusion polypeptides, antigenic peptides
and **antibodies**.

L19 ANSWER 20 OF 178 USPATFULL
AN 2001:212420 USPATFULL
TI Immunostimulatory nucleic acids for inducing a Th2 immune response
IN McCluskie, Michael J., Ottawa, Canada
Davis, Heather L., Ottawa, Canada
PI US 2001044416 A1 20011122
AI US 2001-768012 A1 20010122 (9)
PRAI US 2000-177461 20000120 (60)
DT Utility

FS APPLICATION
LREP Helen Lockhart, c/o Wolf, Greenfield & Sacks, P.C., Federal Reserve
Plaza, 600 Atlantic Avenue, Boston, MA, 02210-2211
CLMN Number of Claims: 153
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 3831
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to methods and products for inducing an immune
response using immunostimulatory nucleic acids. In particular the
immunostimulatory nucleic acids preferentially induce a Th2 immune
response. The invention is useful for treating and preventing disorders
associated with a Th1 immune response or for creating a Th2 environment
for treating disorders that are sensitive to Th2 immune responses.

L19 ANSWER 21 OF 178 USPATFULL
AN 2001:208887 USPATFULL
TI Aromatic heterocyclic compound as antiinflammatory agents
IN Cirillo, Pier F., Woodbury, CT, United States
Gilmore, Thomas A., Middlebury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Regan, John R., Larchmont, NY, United States
Zhang, Lin-Hua, New Fairfield, CT, United States
PA Boerhinger Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United
States (U.S. corporation)
PI US 6319921 B1 20011120
AI US 2000-484638 20000118 (9)
PRAI US 1999-116400 19990119 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Ramsuer, Robert W.
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2297
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel aromatic heterocyclic compounds of the formula (I)
wherein Ar.sub.1, Ar.sub.2, L, Q and X are described herein. The
compounds are useful in pharmaceutic compositions for treating diseases
or pathological conditions involving inflammation such as chronic
inflammatory diseases. Also disclosed are processes of making such
compounds. ##STR1##

L19 ANSWER 22 OF 178 USPATFULL
AN 2001:202682 USPATFULL

TI Therapeutic methods employing disulfide derivatives of dithiocarbonates
and compositions useful therefor
IN Lai, Ching-San, Encinitas, CA, United States
Vassilev, Vassil, San Diego, CA, United States
PA Medinox, Inc., San Diego, CA, United States (U.S. corporation)
PI US 6316502 B1 20011113
AI US 2000-565666 20000505 (9)
RLI Division of Ser. No. US 1998-103639, filed on 23 Jun 1998, now patented,
Pat. No. US 6093743
DT Utility
FS GRANTED
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Reiter, Stephen E.Foley & Lardner
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2591
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a novel dithiocarbamamte disulfide dimer
useful in various therapeutic treatments, either alone or in combination
with other active agents. In one method, the disulfide derivative of a
dithiocarbamate is coadministered with an agent that inactivates (or
inhibits the production of) species that induce the expression of nitric
oxide synthase to reduce the production of such species, while, at the
same time reducing nitric oxide levels in the subject. In another
embodiment, free iron ion levels are reduced in a subject by
administration of a disulfide derivative of a dithiocarbamate(s) to
scavenge free iron ions, for example, in subjects undergoing
anthracycline chemotherapy. In another embodiment, cyanide levels are
reduced in a subject by administration of a disulfide derivative of a
dithiocarbamate so as to bind cyanide in the subject. In a further
aspect, the present invention relates to compositions and formulations
useful in such therapeutic methods.

L19 ANSWER 23 OF 178 USPATFULL
AN 2001:202603 USPATFULL
TI DNA cytokine vaccines and use of same for protective immunity against
multiple **sclerosis**
IN Karin, Nathan, Haifa, Israel
Youssef, Sawsan, Rama Villag, Israel
Wildbaum, Gizi, Kiriat-Yam, Israel
PA Technion Research and Development Foundation LTD., Haifa, Israel
(non-U.S. corporation)

PI US 6316420 B1 20011113
AI US 1998-123485 19980728 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Priebe, Scott D.; Assistant Examiner: Beckerleg, Anne
Marie S.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 36 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 1743
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for treating a mammal for inducing protective immunity against an autoimmune disease including the step of administering to the mammal a therapeutic composition including a recombinant construct including an isolated nucleic acid sequence encoding a cytokine, the nucleic acid sequence being operatively linked to one or more transcription control sequences. A method for treating a mammal for inducing protective immunity against an autoimmune disease including the steps of (a) removing cells of the mammal; (b) transducing the cells in vitro with a recombinant construct including an isolated nucleic acid sequence encoding a cytokine, the nucleic acid sequence being operatively linked to one or more transcription control sequences; and (c) reintroducing the transduced cells to the mammal. A pharmaceutical composition including (a) a recombinant construct including an isolated nucleic acid sequence encoding a cytokine, the nucleic acid sequence being operatively linked to one or more transcription control sequences; and (b) a pharmaceutically acceptable carrier. And an **antibody** raised against a cytokine expressed by cells transduced with a recombinant construct including an isolated nucleic acid sequence encoding the cytokine, the nucleic acid sequence being operatively linked to one or more transcription control sequences.

L19 ANSWER 24 OF 178 USPATFULL
AN 2001:200228 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Collins-Racie, Lisa A., Acton, MA, United States
Evans, Cheryl, Germantown, MD, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Co. Dublin, Ireland

Agostino, Michael J., Andover, MA, United States
Steininger, Robert J., II, Cambridge, MA, United States
Spaulding, Vikki, Lowell, MA, United States
Wong, Gordon G., Brookline, MA, United States
Clark, Hilary, So. San Francisco, CA, United States
Fechtel, Kim, Arlington, MA, United States
PI US 2001039335 A1 20011108
AI US 2000-729674 A1 20001204 (9)
RLI Continuation of Ser. No. US 2000-539330, filed on 30 Mar 2000, PENDING
Continuation-in-part of Ser. No. US 1998-197886, filed on 23 Nov 1998,
ABANDONED
PRAI US 1997-126425 19970410 (60)
US 1997-67454 19971204 (60)
US 1997-68379 19971220 (60)
US 1998-70346 19980102 (60)
US 1998-70643 19980107 (60)
US 1998-70755 19980108 (60)
US 1998-71304 19980113 (60)
US 1998-72134 19980122 (60)
US 1998-73095 19980130 (60)
US 1998-75038 19980218 (60)
DT Utility
FS APPLICATION
LREP Alice O. Carroll, Esq., HAMILTON, BROOK, SMITH & REYNOLDS, P.C., Two
Militia Drive, Lexington, MA, 02421-4799
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Page(s)
LN.CNT 18073
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 25 OF 178 USPATFULL
AN 2001:200183 USPATFULL
TI Aromatic heterocyclic compounds and their use as anti-inflammatory
agents
IN Regan, John R., Larchmont, NY, United States
Hickey, Eugene R., Danbury, CT, United States
Moss, Neil, Ridgefield, CT, United States
Cywin, Charles L., Bethel, CT, United States
Pargellis, Christopher, West Redding, CT, United States
Gilmore, Thomas A., Middlebury, CT, United States
PI US 2001039290 A1 20011108
AI US 2001-808084 A1 20010314 (9)
RLI Division of Ser. No. US 1999-461446, filed on 14 Dec 1999, GRANTED, Pat.
No. US 6228881 Division of Ser. No. US 1998-181743, filed on 29 Oct
1998, GRANTED, Pat. No. US 6080763
PRAI US 1997-64102 19971103 (60)
DT Utility
FS APPLICATION
LREP BOEHRINGER INGELHEIM CORPORATION, 900 RIDGEBURY ROAD, P O BOX 368,

RIDGEFIELD, CT, 06877
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2147
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel aromatic heterocyclic compounds inhibit cytokines production involved in immunoregulation and inflammation such as interleukin-1 and tumor necrosis factor production. The compounds are therefore useful in pharmaceutic compositions for treating diseases or pathological conditions involving inflammation such as chronic inflammatory diseases.

L19 ANSWER 26 OF 178 USPATFULL
AN 2001:196824 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Racie, Lisa A., Acton, MA, United States
Evans, Cheryl, Germantown, MD, United States
Merberg, David, Acton, MA, United States
Mi, Sha, Belmont, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6312921 B1 20011106
AI US 1998-175928 19981020 (9)
RLI Continuation-in-part of Ser. No. US 1998-80478, filed on 18 May 1998, now abandoned Continuation-in-part of Ser. No. US 1997-976110, filed on 21 Nov 1997, now abandoned Continuation-in-part of Ser. No. US 1996-686878, filed on 26 Jul 1996, now patented, Pat. No. US 5708157 Continuation-in-part of Ser. No. US 1996-702081, filed on 23 Aug 1996, now abandoned Continuation-in-part of Ser. No. US 1996-721489, filed on 27 Sep 1996, now patented, Pat. No. US 5786465 Continuation-in-part of Ser. No. US 1996-721924, filed on 27 Sep 1996, now patented, Pat. No. US 5969125 Continuation-in-part of Ser. No. US 1996-686878, filed on 26 Jul 1996, now patented, Pat. No. US 5708157
DT Utility
FS GRANTED
EXNAM Primary Examiner: Carlson, Karen Cochrane; Assistant Examiner: Mitra, Rita
LREP Lahive & Cockfield, LLP, Mandragouras, Amy E., Milasincic, Debra J.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 15 Drawing Page(s)

LN.CNT 3531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 27 OF 178 USPATFULL
AN 2001:196641 USPATFULL
TI Methods for production of the oxidized glutathione composite with
cis-diamminedichloroplatinum and pharmaceutical compositions based
thereof regulating metabolism, proliferation, differentiation and
apoptotic mechanisms for normal and transformed cells
IN Kozhemyakin, Leonid A., St. Petersburg, Russian Federation
Balasovski, Mark B., St. Petersburg, Russian Federation
PA Novelos Therapeutics, Inc., Newton, MA, United States (U.S. corporation)
PI US 6312734 B1 20011106
AI US 1999-241232 19990201 (9)
RLI Continuation-in-part of Ser. No. US 1999-237801, filed on 27 Jan 1999,
now abandoned
PRAI RU 1998-120753 19981123
DT Utility
FS GRANTED
EXNAM Primary Examiner: Russel, Jeffrey E.
LREP Wolf, Greenfield & Sacks, P.C.
CLMN Number of Claims: 136
ECL Exemplary Claim: 70
DRWN 47 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 4627
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a composite for the **treatment** of a variety of medical conditions, the composite comprising an oxidized
glutathione-based compound, which has a disulfide bond, and a metal
material, in particular where the metal is either platinum or palladium.
The oxidized glutathione-based compound and metal material can be
present in a ratio of 3000 to 1 and preferably 1000 to 1. The oxidized
glutathione-based compound can be oxidized glutathione itself or salts
or derivatives. A feature of the invention is that the composite has a
more stabilized disulfide bond than the oxidized glutathione-based
compound itself. Methods for preparing the composite are provided, such
methods being beneficial in that the composite is provided in high
yields and at high purity. Methods for treating various medical
conditions with the composites of the present invention are also
disclosed.

L19 ANSWER 28 OF 178 USPATFULL
AN 2001:196603 USPATFULL
TI Cancer **treatment** methods using therapeutic conjugates that bind to aminophospholipids
IN Thorpe, Philip E., Dallas, TX, United States
Ran, Sophia, Dallas, TX, United States
PA Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)
PI US 6312694 B1 20011106
AI US 1999-351457 19990712 (9)
PRAI US 1998-92589 19980713 (60)
US 1998-110600 19981202 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Williams, Morgan & Amerson
CLMN Number of Claims: 50
ECL Exemplary Claim: 1,2,3,4
DRWN 6 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 8243
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is the surprising discovery that aminophospholipids, such as phosphatidylserine and phosphatidylethanolaminie, are specific, accessible and stable markers of the luminal surface of tumor blood vessels. The present invention thus provides aminophospholipid-targeted diagnostic and therapeutic constructs for use in tumor intervention.

Antibody-therapeutic agent conjugates and constructs that bind to aminophospholipids are particularly provided, as are methods of specifically delivering therapeutic agents, including toxins and coagulants, to the stably-expressed aminophospholipids of tumor blood vessels, thereby inducing thrombosis, necrosis and tumor regression.

L19 ANSWER 29 OF 178 USPATFULL
AN 2001:182105 USPATFULL
TI Controlled delivery of antigens
IN Caplan, Michael, Woodbridge, CT, United States
Bannon, Gary A., Little Rock, AR, United States
Burks, A. Wesley, JR., Little Rock, AR, United States
Sampson, Hugh A., Larchmont, NY, United States
PI US 2001031262 A1 20011018
AI US 2000-730921 A1 20001206 (9)
PRAI US 1999-169330 19991206 (60)
DT Utility
FS APPLICATION
LREP Patrea L. Pabst, Arnall Golden & Gregory, LLP, 2800 One Atlantic Center, 1201 West Peachtree Street, Atlanta, GA, 30309-3450
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN No Drawings

LN.CNT 1143
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Formulations and methods have been developed for delivering antigens to individuals in a manner that substantially reduces contact between the antigen and IgE receptors displayed on the surfaces of cells involved in mediating allergic responses, which target delivery of antigen to dendritic and other phagocytic APCs, and which have improved pharmacokinetics. By reducing direct and indirect association of antigens with antigen-specific IgE **antibodies**, the risk of an allergic reaction, possibly anaphylatic shock, is reduced or eliminated.
Particularly preferred antigens are those that may elicit anaphylaxis in individuals, including food antigens, insect venom and rubber-related antigens. In the preferred embodiments, the compositions include one or more antigens in a delivery material such as a polymer, in the form of particles or a gel, or lipid vesicles or liposomes, any of which can be stabilized or targeted to enhance delivery. Preferably, the antigen is surrounded by the encapsulation material. Alternatively or additionally, the antigen is displayed on the surface of the encapsulation material.
One result of encapsulating antigen is the reduction in association with antigen-specific IgE **antibodies**. In some embodiments, antigens are stabilized or protected from degradation until the antigen can be recognized and endocytized by APCs which are involved in elicting cellular and humoral immune responses. In a preferred embodiment, the formulation is designed to deliver antigens to individuals in a manner designed to promote a Th1-type mediated immune response and/or in a manner designed to suppress a Th2 response. In still another embodiment, the formulation effects preferential release of the antigen within APCs.

L19 ANSWER 30 OF 178 USPATFULL
AN 2001:182096 USPATFULL
TI Autologous immune cell **therapy**: cell compositions, methods and applications to **treatment** of human disease
IN Gruenberg, Micheal L., Poway, CA, United States
PI US 2001031253 A1 20011018
AI US 2001-824906 A1 20010402 (9)
RLI Division of Ser. No. US 1996-700565, filed on 25 Jul 1996, PENDING

Division of Ser. No. WO 1996-US12170, filed on 24 Jul 1996, UNKNOWN
PRAI US 1995-44693 19950726 (60)
DT Utility
FS APPLICATION
LREP Stephanie Seidman, Heller Ehrman White & McAuliffe LLP, 4250 Executive
Square, 7th Floor, La Jolla, CA, 92037
CLMN Number of Claims: 101
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2692
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions containing clinically relevant numbers of immune cells that
have been isolated from a patient differentiated and/or expanded ex
vivo. Methods for treating or preventing disease or otherwise altering
the immune status of the patient by reinfusing such cells into the donor
are also provided. Methods for expanding and/or immune cells, including
effector cells, in the absence of exogenous IL-2, and for administering
the cells in the absence of co-infused IL-2 are also provided.

L19 ANSWER 31 OF 178 USPATFULL
AN 2001:179242 USPATFULL
TI Therapeutic multispecific compounds comprised of anti-FCA receptor
antibodies
IN Deo, Yashwant M., Audubon, PA, United States
Graziano, Robert, Frenchtown, NJ, United States
Keler, Tibor, Ottsville, PA, United States
PA Medarex, Inc., Princeton, NJ, United States (U.S. corporation)
PI US 6303755 B1 20011016
AI US 1999-262724 19990304 (9)
RLI Continuation of Ser. No. US 1996-678194, filed on 11 Jul 1996, now
patented, Pat. No. US 5922845
DT Utility
FS GRANTED
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Lahive & Cockfield, LLP, Remillard, Jane E., Dini, Peter W.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1,17
DRWN 18 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 2050
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor
antibodies and methods of use are provided.

L19 ANSWER 32 OF 178 USPATFULL
AN 2001:170889 USPATFULL
TI Monocyte-derived dendritic cell subsets
IN Punnonen, Juha, Palo Alto, CA, United States
Chang, Chia-Chun J., Los Gatos, CA, United States

PI US 2001026937 A1 20011004
AI US 2001-760388 A1 20010110 (9)
PRAI US 2000-175552 20000111 (60)
US 2000-181957 20000210 (60)
DT Utility
FS APPLICATION
LREP LAW OFFICES OF JONATHAN ALAN QUINE, P O BOX 458, ALAMEDA, CA, 94501
CLMN Number of Claims: 69
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 3189
AB A novel subset of monocyte-derived dendritic cells are provided. Methods for producing these monocyte-derived dendritic cells and compositions comprising the dendritic cells of the invention are also provided. Methods for inducing an immune response to an antigen of interest using the dendritic cells of the invention are provided. Also provided are methods for therapeutically or prophylactically treating a disease in a subject suffering from the disease using the dendritic cells.

L19 ANSWER 33 OF 178 USPATFULL
AN 2001:168261 USPATFULL
TI Aromatic heterocyclic compounds as anti-inflammatory agents
IN Cirillo, Pier F., Woodbury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Regan, John R., Larchmont, NY, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United States (U.S. corporation)
PI US 6297381 B1 20011002
AI US 2000-503385 20000214 (9)
PRAI US 1999-124147 19990312 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Shah, Mukund J.; Assistant Examiner: Patel, Sudhaker B.
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1389
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel aromatic heterocyclic compounds of the formula(I) wherein Ar.sub.1,Ar.sub.2,L,Q and X are described herein. The compounds are useful in pharmaceutic compositions for treating diseases or pathological conditions. Also disclosed are processes of making such compounds. ##STR1##

L19 ANSWER 34 OF 178 USPATFULL
AN 2001:163320 USPATFULL
TI Anti-interleukin-1 receptor **antagonist antibodies** and uses thereof
IN Ford, John, San Mateo, CA, United States
Pace, Ann, Scotts Valley, CA, United States
PA Hyseq, Inc., Sunnyvale, CA, United States (U.S. corporation)
PI US 6294655 B1 20010925
AI US 1999-417455 19991013 (9)
RLI Continuation-in-part of Ser. No. US 1999-348942, filed on 7 Jul 1999
Continuation of Ser. No. US 1999-287210, filed on 5 Apr 1999, now
abandoned Continuation-in-part of Ser. No. US 1999-251370, filed on 17
Feb 1999, now abandoned Continuation-in-part of Ser. No. US 1998-127698,
filed on 31 Jul 1998, now abandoned Continuation-in-part of Ser. No. US
1999-229591, filed on 13 Jan 1999, now abandoned Continuation of Ser.
No. US 1998-99818, filed on 19 Jun 1998, now abandoned , said Ser. No.
US 127698 Continuation-in-part of Ser. No. US 1998-82364, filed on 20
May 1998, now abandoned , said Ser. No. US 99818 Continuation-in-part of
Ser. No. US 1998-82364, filed on 20 May 1998, now abandoned
Continuation-in-part of Ser. No. US 1998-79909, filed on 15 May 1998,
now abandoned Continuation-in-part of Ser. No. US 1998-55010, filed on 3
Apr 1998, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Spector, Lorraine
LREP Marshall, O'Toole Gerstein, Murray & Borun
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 4656
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel nucleic acids, the novel polypeptide sequences encoded by these nucleic acids and uses thereof.
These novel polynucleotide and polypeptide sequences were determined to
be a novel Interleukin-1 Receptor **Antagonist**. Also provided are **antibodies** which bind the **antagonist**, methods of detecting the **antagonist**, and kits containing the **antibodies**.

L19 ANSWER 35 OF 178 USPATFULL
AN 2001:163020 USPATFULL
TI Methods for treating fibroproliferative diseases
IN Peterson, Theresa C., Nova Scotia, Canada
PA Dalhousie University, Halifax, Canada (non-U.S. corporation)
PI US 6294350 B1 20010925
AI US 1999-433621 19991102 (9)

RLI Continuation-in-part of Ser. No. US 1998-92317, filed on 5 Jun 1998, now
patented, Pat. No. US 6025151 Continuation-in-part of Ser. No. US
1997-870096, filed on 5 Jun 1997, now patented, Pat. No. US 5985592
DT Utility
FS GRANTED
EXNAM Primary Examiner: Leary, Louise N.
LREP Foley & Lardner, Reiter, Stephen E.
CLMN Number of Claims: 42
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 1148
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In accordance with the present invention, fibroproliferative disease or
condition characterized by such symptoms as increased levels of c-Jun
homodimers, increased heterodimerization of c-Jun with another signaling
peptide, increased levels of phosphorylated c-Jun, or increased presence
of Jun kinase are treated by administering to the subject an amount of a
compound effective to ameliorate one or more of the symptoms of the
disease or condition, for example, an antiproliferative or antifibrotic
agent. Preferred compounds for administration according to the invention
are antisense c-Jun oligonucleotides and compounds that block c-Jun
phosphorylation, such as pentoxifylline, or a functional derivative or
metabolite thereof. Also provided by the present invention are in vitro
tests for identifying whether a test compound is useful for
treatment of a subject afflicted with such a disease and kits
useful for conducting such assays.

L19 ANSWER 36 OF 178 USPATFULL
AN 2001:160802 USPATFULL
TI Interleukins-21 and 22
IN Ebner, Reinhard, Gaithersburg, MD, United States
Ruben, Steven M., Olney, MD, United States
PI US 2001023070 A1 20010920
AI US 2000-731816 A1 20001208 (9)
RLI Continuation-in-part of Ser. No. US 1999-320713, filed on 27 May 1999,
PENDING Continuation-in-part of Ser. No. WO 1999-US11644, filed on 27
May 1999, UNKNOWN
PRAI US 1998-87340 19980529 (60)
US 1999-131965 19990430 (60)
US 1999-169837 19991209 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 7740
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel human proteins designated
Interleukin-21 (IL-21) and Interleukin-22 (IL-22), and isolated
polynucleotides encoding these proteins. Also provided are vectors, host
cells, **antibodies**, and recombinant methods for producing these
human proteins. The invention further relates to diagnostic and
therapeutic methods useful for diagnosing, treating, and/or preventing
disorders related to these novel human proteins.

L19 ANSWER 37 OF 178 USPATFULL
AN 2001:155773 USPATFULL
TI Composition
IN Potter, Barry Victor Lloyd, The Oxford Science Park, Great Britain
Reed, Michael John, The Oxford Science Park, Great Britain
Elger, Walter, Berlin, Germany, Federal Republic of
Reddersen, Gudrun, Jena, Germany, Federal Republic of
Proske, Heinrich-Thomas, Berlin, Germany, Federal Republic of
PI US 2001021707 A1 20010913
AI US 2001-755429 A1 20010105 (9)
PRAI GB 2000-792 20000114
GB 2000-2115 20000128
US 2000-218730 20000717 (60)
DT Utility
FS APPLICATION
LREP FROMMER LAWRENCE & HAUG LLP, 745 Fifth Avenue, New York, NY, 10151
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 2046
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB There is provided a pharmaceutical composition comprising (i) a compound
of the formula ##STR1##

wherein: X is a hydrocarbyl ring having at least 4 atoms in the ring; K
is a hydrocarbyl group; Rs is a sulphamate group; (ii) optionally
admixed with a pharmaceutically acceptable carrier, diluent, excipient
or adjuvant, wherein the compound is present in an amount to provide a
dosage of no greater than 200 .mu.g/day.

L19 ANSWER 38 OF 178 USPATFULL
AN 2001:155766 USPATFULL
TI 49 human secreted proteins
IN Moore, Paul A., Germantown, MD, United States
Ruben, Steven M., Oley, MD, United States
Olsen, Henrik S., Gaithersburg, MD, United States

Shi, Yanggu, Gaithersburg, MD, United States
Rosen, Craig A., Laytonsville, MD, United States
Florence, Kimberly A., Rockville, MD, United States
Soppet, Daniel R., Centreville, VA, United States
Lafleur, David W., Washington, DC, United States
Endress, Gregory A., Potomac, MD, United States
Ebner, Reinhard, Gaithersburg, MD, United States
Komatsoulis, George, Silver Spring, MD, United States
Duan, Roxanne D., Bethesda, MD, United States
PI US 2001021700 A1 20010913
AI US 2000-739254 A1 20001219 (9)
RLI Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000, ABANDONED
Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,
UNKNOWN
PRAI US 1998-97917 19980825 (60)
US 1998-98634 19980831 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 15462
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel human secreted proteins and
isolated nucleic acids containing the coding regions of the genes
encoding such proteins. Also provided are vectors, host cells, **antibodies**, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic
methods useful for diagnosing and treating diseases, disorders, and/or
conditions related to these novel human secreted proteins.

L19 ANSWER 39 OF 178 USPATFULL
AN 2001:155579 USPATFULL
TI Materials and methods for detection and **treatment** of immune system dysfunctions
IN Clare-Salzer, Michael, Gainesville, FL, United States
PI US 2001021510 A1 20010913
AI US 2001-821435 A1 20010329 (9)
RLI Division of Ser. No. US 1999-322628, filed on 28 May 1999, GRANTED, Pat.
No. US 6218133 Division of Ser. No. US 1997-916586, filed on 22 Aug
1997, GRANTED, Pat. No. US 6168792 Continuation-in-part of Ser. No. US
1996-701928, filed on 23 Aug 1996, GRANTED, Pat. No. US 5939069
DT Utility
FS APPLICATION
LREP SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W.
41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
CLMN Number of Claims: 9

ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 813
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The subject invention concerns novel materials and methods for the
treatment and/or prevention of autoimmune disease. In a specific embodiment, elevated production of prostaglandin synthase-2 (PGS-2) is
correlated with autoimmune dysfunction.

L19 ANSWER 40 OF 178 USPATFULL
AN 2001:141921 USPATFULL
TI Immunomodulating compositions from bile
IN Rang, Romeo, Bucharest, Romania
PA Lorus Therapeutics Inc., Ontario, Canada (non-U.S. corporation)
PI US 6280774 B1 20010828
WO 9507089 19950316
AI US 1996-612921 19960516 (8)
WO 1994-CA494 19940909
19960516 PCT 371 date
19960516 PCT 102(e) date
RLI Continuation of Ser. No. US 1994-231726, filed on 4 Apr 1994, now
abandoned Continuation of Ser. No. US 1993-155303, filed on 22 Nov 1993,
now abandoned Continuation of Ser. No. US 1993-118269, filed on 9 Sep
1993, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Witz, Jean C.
LREP McDonnell Boehnen Hulbert & Berghoff
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 21 Drawing Page(s)
LN.CNT 3417
AB The present invention relates to a composition for use as an immunomodulator comprising small molecular weight components of less
than 3000 daltons, and having the following properties: a) is extractable from bile of animals; b) is capable of stimulating monocytes
and macrophages in vitro; c) is capable of modulating tumor necrosis
factor production; d) contains no measurable IL-1a, IL-1b, **TNF**, IL-6, IL-8, IL-4, GM-CSF or IFN-gamma; e) has an anti-proliferative
effect in a malignant mouse hybridoma cell line; f) shows no cytotoxicity to human peripheral blood mononuclear cells; and g) is not
an endotoxin. The invention also relates to a method of preparing the
composition and its use an immunomodulator.

L19 ANSWER 41 OF 178 USPATFULL
AN 2001:141888 USPATFULL
TI Method of inhibiting angiogenesis using secreted proteins
IN Jacobs, Kenneth, Newton, MA, United States

McCoy, John M., Reading, MA, United States
Racie, Lisa A., Acton, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Dublin, Ireland
Evans, Cheryl, Germantown, MD, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6280739 B1 20010828
AI US 1997-885469 19970627 (8)
RLI Continuation-in-part of Ser. No. US 1996-743684, filed on 6 Nov 1996, now abandoned Continuation-in-part of Ser. No. US 1996-634325, filed on 18 Apr 1996, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Carlson, Karen Cochrane; Assistant Examiner: Houze, Thomas A.
LREP Lahive & Cockfield, LLP, Lauro, Peter C., Mandragouras, Amy R.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1678
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel proteins are disclosed.

L19 ANSWER 42 OF 178 USPATFULL
AN 2001:139607 USPATFULL
TI METHOD OF **TREATMENT** WITH A SECRETED PROTIEN
IN JACOBS, KENNETH, NEWTON, MA, United States
MCCOY, JOHN M., READING, MA, United States
RACIE, LISA A., ACTON, MA, United States
LAVALLIE, EDWARD R., HARVARD, MA, United States
TREACY, MAURICE, CHESTNUT HILL, MA, United States
EVANS, CHERYL, WOBURN, MA, United States
AGOSTINO, MICHAEL J., ANDOVER, MA, United States
LU, ZHIJIAN, BEDFORD, MA, United States
MERBERG, DAVID, ACTON, MA, United States
TASHIRO, KEI, KYOTO, Japan
NAKAMURA, TOMOYUKI, SAN DIEGO, CA, United States
HONJO, TAKUKU, KYOTO, Japan
PI US 2001016650 A1 20010823
AI US 1998-83002 A1 19980521 (9)
RLI Continuation-in-part of Ser. No. US 1997-885610, filed on 30 Jun 1997, ABANDONED Continuation-in-part of Ser. No. US 1996-634325, filed on 18 Apr 1996, ABANDONED
DT Utility
FS APPLICATION
LREP LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 2052
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel proteins and methods of **treatment** using sameare

disclosed.

L19 ANSWER 43 OF 178 USPATFULL
AN 2001:139604 USPATFULL
TI 29 human secreted proteins
IN Ruben, Steven M., Olney, MD, United States
Rosen, Craig A., Laytonsville, MD, United States
Fan, Ping, Gaithersburg, MD, United States
Kyaw, Hla, Frederick, MD, United States
Wei, Ying-Fei, Berkeley, CA, United States
PI US 2001016647 A1 20010823
AI US 2000-729835 A1 20001206 (9)
RLI Division of Ser. No. US 1999-257179, filed on 25 Feb 1999, PENDING
Continuation-in-part of Ser. No. WO 1998-US17709, filed on 27 Aug 1998,
UNKNOWN
PRAI US 1997-56270 19970829 (60)
US 1997-56271 19970829 (60)
US 1997-56247 19970829 (60)
US 1997-56073 19970829 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 6098
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel human secreted proteins and
isolated nucleic acids containing the coding regions of the genes
encoding such proteins. Also provided are vectors, host cells, **antibodies**, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic
methods useful for diagnosing and treating disorders related to these
novel human secreted proteins.

L19 ANSWER 44 OF 178 USPATFULL
AN 2001:136440 USPATFULL
TI Use of interleukin-10 to produce a population of suppressor cells
IN Roncarolo, Maria-Grazia, Los Altos, CA, United States
Malefyt, Rene de Waal, Sunnyvale, CA, United States
Bacchetta, Rosa, Milano Due, Italy
Groux, Herve M., Palo Alto, CA, United States
de Vries, Jan E., Los Altos, CA, United States
PA Schering Corporation, Kenilworth, NJ, United States (U.S. corporation)
PI US 6277635 B1 20010821
AI US 1996-643810 19960506 (8)
RLI Continuation-in-part of Ser. No. US 1992-846208, filed on 4 Mar 1992,
now abandoned
DT Utility

FS GRANTED
EXNAM Primary Examiner: Allen, Marianne P.
LREP Wang, Hugh, Ching, Edwin P., Apple, Ted
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 28 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 2584
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Interleukin-10 for producing a population of cells which are capable of
inhibiting or suppressing reactions to alloantigens, for example in
graft-versus-host disease or tissue rejection, is described.
Interleukin-10 for reducing responses in mixed lymphocyte response (MLR)
is also described. Exogenous or induced endogenous IL-10 may be used for
the inhibition or suppression of the reactions to alloantigens.

L19 ANSWER 45 OF 178 USPATFULL
AN 2001:133879 USPATFULL
TI Therapeutic multispecific compounds comprised of anti-Fcalpha receptor
antibodies
IN Deo, Yashwant M., Audubon, PA, United States
Graziano, Robert, Frenchtown, NJ, United States
Keler, Tibor, Ottsville, PA, United States
PA Mederax, Inc. (U.S. corporation)
PI US 2001014328 A1 20010816
AI US 2001-772120 A1 20010126 (9)
RLI Continuation of Ser. No. US 1997-890011, filed on 10 Jul 1997, GRANTED,
Pat. No. US 6193966 Continuation-in-part of Ser. No. US 1996-678194,
filed on 11 Jul 1996, GRANTED, Pat. No. US 5922845
DT Utility
FS APPLICATION
LREP LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
CLMN Number of Claims: 68
ECL Exemplary Claim: 1
DRWN 28 Drawing Page(s)
LN.CNT 2753
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor
antibodies and methods of use are provided.

L19 ANSWER 46 OF 178 USPATFULL
AN 2001:131349 USPATFULL
TI Alkylated resorcinol derivatives for the **treatment** of immune diseases
IN Travis, Craig A., South Miami, FL, United States
PA Immugen Pharmaceuticals Inc., Miami, FL, United States (U.S. corporation)
PI US 6274635 B1 20010814
AI US 2000-533386 20000322 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Padmanabhan, Sreeni; Assistant Examiner: Price, Elvis

O.
LREP Leydig, Voit & Mayer Ltd.
CLMN Number of Claims: 56
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1897
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method, compounds, and compositions for treating a disease associated with immune dysfunction. In accordance with the method, a pharmacologically-acceptable composition including at least one compound selected from the group of compounds consisting of 5-alkyl-resorcinol derivatives, cannabinol derivatives, cannabidiol derivatives, cannabigerol derivatives, and combinations thereof is administered to a patient under conditions sufficient to attenuate the dysfunction within the immune system. The invention also provides an antiviral cannabinol derivative that can be used in the inventive method. The invention also provides an alkylated resorcinol derivative and a method of using the alkylated resorcinol derivative to attenuate the growth of a neoplasm. The method and compound are useful for treating diseases of the immune system, such as HIV disease and neoplastic disorders.

L19 ANSWER 47 OF 178 USPATFULL
AN 2001:128901 USPATFULL
TI 36 human secreted proteins
IN LaFleur, David W., Washington, DC, United States
Soppet, Daniel R., Centreville, VA, United States
Olsen, Henrik, Gaithersburg, MD, United States
Ruben, Steven M., Olney, MD, United States
Ni, Jian, Rockville, MD, United States
Rosen, Craig A., Laytonsville, MD, United States
Brewer, Laurie A., St. Paul, MN, United States
Duan, Roxanne, Bethesda, MD, United States
Ebner, Reinhard, Gaithersburg, MD, United States
PI US 2001012889 A1 20010809
AI US 2000-739907 A1 20001220 (9)
RLI Continuation of Ser. No. US 1999-348457, filed on 7 Jul 1999, ABANDONED
Continuation-in-part of Ser. No. WO 1999-US108, filed on 6 Jan 1999, UNKNOWN
PRAI US 1998-70704 19980107 (60)
US 1998-70658 19980107 (60)
US 1998-70692 19980107 (60)
US 1998-70657 19980107 (60)
DT Utility
FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 10341
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to 36 novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, **antibodies**, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating disorders related to these novel human secreted proteins.

L19 ANSWER 48 OF 178 USPATFULL
AN 2001:123426 USPATFULL
TI PROSTATE DERIVED ETS FACTOR
IN LIBERMANN, TOWIA ARON, NEWTON, MA, United States
OETTGEN, JOERG PETER, BROOKLINE, MA, United States
KUNSCH, CHARLES A., NORCROSS, GA, United States
ENDRESS, GREGORY A., POTOMAC, MD, United States
ROSEN, CRAIG A., LAYTONSVILLE, MD, United States
PI US 2001010934 A1 20010802
AI US 1998-126945 A1 19980731 (9)
DT Utility
FS APPLICATION
LREP STERNE KESSLER GOLDSTEIN AND FOX, SUITE 600, 1100 NEW YORK AVENUE N W, WASHINGTON, DC, 200053934
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 4218
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a novel human protein called Prostate Derived Ets Factor, and isolated polynucleotides encoding this protein. Also provided are vectors, host cells, **antibodies**, and recombinant methods for producing this human protein. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating disorders related to this novel human protein.

L19 ANSWER 49 OF 178 USPATFULL
AN 2001:119059 USPATFULL
TI Immunomodulating compositions for **treatment** of immune system disorders
IN Rang, Romeo G., Bucharest, Romania
Percheson, Paul B., Ontario, Canada
PI US 2001009680 A1 20010726
AI US 2001-764010 A1 20010117 (9)

RLI Continuation of Ser. No. US 1995-404932, filed on 16 Mar 1995, ABANDONED
DT Utility
FS APPLICATION
LREP MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE
3200, CHICAGO, IL, 60606
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3900
AB The present invention relates to a composition for use as an immunomodulator comprising small molecular weight components of less than 3000 daltons, and having the following properties: a) is extractable from bile of animals; b) is capable of stimulating monocytes and macrophages in vitro and in vivo; c) is capable of modulating tumor necrosis factor production; d) contains no measurable IL-1.alpha., IL-1.beta., **TNF**, IL-6, IL-8, IL-4, GM-CSF or IFN-.gamma.; e) has an anti-proliferative effect in a malignant mouse hybridoma cell line; f) shows no cytotoxicity to human peripheral blood mononuclear cells or lymphocytes; and g) is not an endotoxin. The invention also relates to a method of preparing the composition, its use as an immunomodulator, and its use in the **treatment** of diseases and conditions having an immunological component.

L19 ANSWER 50 OF 178 USPATFULL
AN 2001:108030 USPATFULL
TI Inhibitors of Interleukin-1.beta. converting enzyme
IN Batchelor, Mark James, Cumnor Hill, United Kingdom
Bebbington, David, Pewsey, United Kingdom
Bemis, Guy W., Arlington, MA, United States
Fridman, Wolf Herman, Paris, France
Gillespie, Roger John, Oaksey, United Kingdom
Golec, Julian M. C., Ashbury, United Kingdom
Gu, Yong, Brookline, MA, United States
Lauffer, David J., Stow, MA, United States
Livingston, David J., Newtonville, MA, United States
Matharu, Saroop Singh, Cricklade, United Kingdom
Mullican, Michael D., Needham, MA, United States
Murcko, Mark A., Holliston, MA, United States
Murdoch, Robert, Highworth, United Kingdom
Nyce, Philip, Milbury, MA, United States
Robidoux, Andrea L. C., Andover, MA, United States
Su, Michael, Newton, MA, United States
Wannamaker, M. Woods, Stow, MA, United States
Wilson, Keith P., Hopkinton, MA, United States
Zelle, Robert E., Stow, MA, United States
PA Vertex Pharmaceuticals, Incorporated, Cambridge, MA, United States (U.S. corporation)
PI US 6258948 B1 20010710
AI US 1999-400639 19990921 (9)

RLI Division of Ser. No. US 1996-761483, filed on 6 Dec 1996
Continuation-in-part of Ser. No. US 1996-712878, filed on 12 Sep 1996,
now patented, Pat. No. US 5985863 Continuation-in-part of Ser. No. US
1996-598332, filed on 8 Feb 1996, now patented, Pat. No. US 5874424
Continuation-in-part of Ser. No. US 1995-575641, filed on 20 Dec 1995,
now patented, Pat. No. US 6008217
PRAI US 1996-31495 19961126 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Kifle, Bruck
LREP Fish & Neave, Haley, Jr., Esq., James F., Joslyn, Kristin M.
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 11 Drawing Page(s)
LN.CNT 13229
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel classes of compounds which are
inhibitors of interleukin-IB converting enzyme. The ICE inhibitors of
this invention are characterized by specific structural and
physicochemical features. This invention also relates to pharmaceutical
compositions comprising these compounds. The compounds and
pharmaceutical compositions of this invention are particularly well
suited for inhibiting ICE activity and consequently, may be
advantageously used as agents against IL-1-, apoptosis-, IGIF-, and
IFN-.gamma.-mediated diseases, inflammatory diseases, autoimmune
diseases, destructive bone disorders, proliferative disorders,
infectious diseases, degenerative diseases, and necrotic diseases. This
invention also relates to methods for inhibiting ICE activity, for
treating interleukin-1-, apoptosis-, IGIF- and IFN-.gamma.-mediated
diseases and decreasing IGIF and IFN-.gamma. production using the
compounds and compositions of this invention. This invention also
relates to methods for preparing N-acylamino compounds.

L19 ANSWER 51 OF 178 USPATFULL
AN 2001:107647 USPATFULL
TI Human **antibodies** that bind human **TNF**.alpha.
IN Salfeld, Jochen G., North Grafton, MA, United States
Allen, Deborah J., Cambridge, United Kingdom
Hoogenboom, Hendricus R. J. M., Hertogsingel, MA, United States
Kaymakcalan, Zehra, Westboro, MA, United States
Labkovsky, Boris, Framingham, MA, United States
Mankovich, John A., Andover, MA, United States
McGuinness, Brian T., Comberton, United Kingdom
Roberts, Andrew J., Cambridge, United Kingdom

Sakorafas, Paul, Newton, MA, United States
Schoenhaut, David, Garfield, NJ, United States
Vaughan, Tristan J., Impington, United Kingdom
White, Michael, Framingham, MA, United States
Wilton, Alison J., Cambridge, United Kingdom
PA BASF Aktiengesellschaft, Rheiland-Pfalz, Germany, Federal Republic of
(non-U.S. corporation)
PI US 6258562 B1 20010710
WO 9729131 19970814
AI US 1999-125098 19990316 (9)
WO 1997-US2219 19970210
19990316 PCT 371 date
19990316 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1996-599226, filed on 9 Feb 1996,
now patented, Pat. No. US 6090382
PRAI US 1996-31476 19961125 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Saunders, David
LREP Lahive & Cockfield, LLP, DeConti, Jr., Giulio A., Hanley, Elizabeth A.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 11 Drawing Page(s)
LN.CNT 2754
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Human **antibodies**, preferably recombinant human **antibodies**, that specifically bind to human tumor necrosis factor .alpha.(hTNF.alpha.) are disclosed. These **antibodies** have high affinity for hTNF.alpha. (e.g., K.sub.d =10.sup.-8 M or less), a slow off rate for hTNF.alpha. dissociation (e.g., K.sub.off =10.sup.-3 sec.sup.-1 or less) and neutralize hTNF.alpha. activity in vitro and in vivo. An **antibody** of the invention can be a full-length **antibody** or an antigen-binding portion thereof. The **antibodies**, or **antibody** portions, of the invention are useful for detecting hTNF.alpha. and for inhibiting hTNF.alpha. activity, e.g., in a human subject suffering from a disorder in which hTNF.alpha. activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human **antibodies** of the invention, and methods of synthesizing the recombinant human **antibodies**, are also encompassed by the invention.

L19 ANSWER 52 OF 178 USPATFULL
AN 2001:105354 USPATFULL
TI 1-OXO- AND 1,3-DIOXOISOINDOLINES AND METHOD OF REDUCING INFLAMMATORY CYTOKINE LEVELS
IN MAN, HON-WAH, NESHANIC STATION, NJ, United States
MULLER, GEORGE W., BRIDGEWATER, NJ, United States
PI US 2001006973 A1 20010705
AI US 1999-270411 A1 19990316 (9)
PRAI US 1998-78180 19980316 (60)

DT Utility
FS APPLICATION
LREP BRUCE M COLLINS, MATHEWS COLLINS SHEPHERD & GOULD, 100 THANET CIRCLE,
SUITE 306, PRINCETON, NJ, 08540
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 707
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB 1-Oxo- and 1,3-dioxo-2-(2,6-dioxopiperidin-3-yl)isoindolines substituted
in the 4- and/or 7-position of the isoindoline ring and optionally
further substituted in the 3-position of the 2,6-dioxopiperidine ring
reduce the levels of inflammatory cytokines such as **TNF**.alpha.
in a mammal. A typical embodiment is 1,3-dioxo-2-(2,6-dioxopiperidin-3-
yl)-4-methylisoindoline

L19 ANSWER 53 OF 178 USPATFULL
AN 2001:105331 USPATFULL
TI GENETIC VACCINE VECTOR ENGINEERING
IN PUNNONEN, JUHA, PALO ALTO, CA, United States
STEMMER, WILLEM P.C., LOS GATOS, CA, United States
WHALEN, ROBERT G., PARIS, France
HOWARD, RUSSELL, LOS ALTOS HILLS, CA, United States
PI US 2001006950 A1 20010705
AI US 1999-247888 A1 19990210 (9)
PRAI US 1998-74294 19980211 (60)
DT Utility
FS APPLICATION
LREP LAW OFFICES OF JONATHAN ALAN QUINE, P O BOX 458, ALAMEDA, CA, 94501
CLMN Number of Claims: 73
ECL Exemplary Claim: 1
DRWN 20 Drawing Page(s)
LN.CNT 4612
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides methods of obtaining improved genetic vaccines
by use of DNA shuffling. Through use of the claimed methods, vectors can
be obtained which exhibit increased efficacy for use as genetic
vaccines. Improved vectors obtained by using the methods can have, for
example, enhanced antigen expression, increased uptake into a cell,
increased stability in a cell, ability to tailor an immune response, and
the like.

L19 ANSWER 54 OF 178 USPATFULL
AN 2001:97961 USPATFULL
TI Inflammatory cell inhibitors
IN Harris, Stephen John, Cowley, United Kingdom
Corkill, Dominic John, Cowley, United Kingdom
PA British Biotech Pharmaceuticals Ltd., Oxford, United Kingdom (non-U.S.

corporation)
PI US 6251940 B1 20010626
WO 9944602 19990910
AI US 1999-355002 19990721 (9)
WO 1999-GB663 19990305
19990721 PCT 371 date
19990721 PCT 102(e) date
PRAI GB 1998-4777 19980307
DT Utility
FS GRANTED
EXNAM Primary Examiner: Reamer, James H.
LREP Greenberg Traurig LLP
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1217
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compounds of general formula (I) ##STR1##

wherein R.sub.4 is an ester or thioester group and R, R.sub.1, R.sub.2,
and R.sub.3 are as specified in the description, inhibit monocyte and/or
macrophage and/or lymphocyte activation and lymphocyte proliferation.

L19 ANSWER 55 OF 178 USPATFULL
AN 2001:97948 USPATFULL
TI Oxyiminoalkanoic acid derivatives with hypoglycemic and hypolipidemic
activity
IN Momose, Yu, Takarazuka, Japan
Odaka, Hiroyuki, Kobe, Japan
Imoto, Hiroshi, Kusatsu, Japan
Kimura, Hiroyuki, Sakai, Japan
Sakamoto, Junichi, Toyonaka, Japan
PA Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)
PI US 6251926 B1 20010626
WO 9958510 19991118
AI US 1999-423854 19991115 (9)
WO 1999-JP2407 19990510
19991115 PCT 371 date
19991115 PCT 102(e) date
PRAI JP 1998-127921 19980511
JP 1998-127922 19980511
DT Utility
FS GRANTED
EXNAM Primary Examiner: Powers, Fiona T.; Assistant Examiner: Wright, Sonya
LREP Riesen, Philippe Y.
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 5841
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides a novel oxyiminoalkanoic acid derivative which
has excellent hypoglycemic and hypolipidemic actions and which is used

for the **treatment** of diabetes mellitus, hyperlipemia, insulin insensitivity, insulin resistance and impaired glucose tolerance.

L19 ANSWER 56 OF 178 USPATFULL
AN 2001:93332 USPATFULL
TI Immunization with plasmid encoding immunogenic proteins and intracellular targeting sequences
IN Williams, William V., Havertown, PA, United States
Madaio, Michael, Bryn Mawr, PA, United States
Weiner, David B., Merion Station, PA, United States
PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
PI US 6248565 B1 20010619
AI US 2000-496301 20000202 (9)
RLI Continuation of Ser. No. US 1997-957001, filed on 23 Oct 1997
PRAI US 1996-29592 19961023 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Park, Hankyel T.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 35
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1952
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Improved vaccines are disclosed. The improved vaccines include a nucleotide sequence that encodes a coding sequence that comprises an immunogenic target protein linked to or comprising an intracellular cellular targeting sequence, the coding sequence being operably linked to regulatory elements are disclosed. Methods of immunizing individuals are disclosed.

L19 ANSWER 57 OF 178 USPATFULL
AN 2001:82778 USPATFULL
TI Polycyclo heterocyclic derivatives as antiinflammatory agents
IN Cirillo, Pier F., Woodbury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Regan, John R., Larchmont, NY, United States
Zhang, Lin-Hua, New Fairfield, CT, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc, Ridgefield, CT, United States (U.S. corporation)
PI US 6242453 B1 20010605
AI US 2000-503263 20000214 (9)
PRAI US 1999-121178 19990222 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Rao, Deepak R.
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 18

ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1136
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel aromatic polycyclo heterocyclic compounds of the formula(I) wherein A, B, C, G, Ar, L, Q and X are described herein. The compounds are useful in pharmaceutical compositions for treating diseases or pathological conditions involving inflammation such as chronic inflammatory disease. Also disclosed are processes of making such compounds. ##STR1##

L19 ANSWER 58 OF 178 USPATFULL
AN 2001:67692 USPATFULL
TI Aromatic heterocyclic compounds and their use as anti-inflammatory agents
IN Regan, John R., Larchmont, NY, United States
Cirillo, Pier F., Woodbury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Moss, Neil, Ridgefield, CT, United States
Cywin, Charles L., Bethel, CT, United States
Pargellis, Christopher, West Redding, CT, United States
Gilmore, Thomas A., Middlebury, CT, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United States (U.S. corporation)
PI US 6228881 B1 20010508
AI US 1999-461446 19991214 (9)
RLI Division of Ser. No. US 1998-181743, filed on 29 Oct 1998
PRAI US 1997-64102 19971103 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Owens, Amelia
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2086
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel aromatic heterocyclic compounds inhibit cytokines production involved in immunoregulation and inflammation such as interleukin-1 and tumor necrosis factor production. The compounds are therefore useful in pharmaceutic compositions for treating diseases or pathological conditions involving inflammation such as chronic inflammatory diseases.

L19 ANSWER 59 OF 178 USPATFULL
AN 2001:67432 USPATFULL
TI Plasmids encoding immunogenic proteins and intracellular targeting sequences

IN Williams, William V., Havertown, PA, United States
Madaio, Michael, Bryn Mawr, PA, United States
Weiner, David B., Merion Station, PA, United States
PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
PI US 6228621 B1 20010508
AI US 1997-957001 19971023 (8)
PRAI US 1996-29592 19961023 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Park, Hankyel
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 40
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1897
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Improved vaccines are disclosed. The improved vaccines include a
nucleotide sequence that encodes a coding sequence that comprises an
immunogenic target protein linked to or comprising an intracellular
cellular targeting sequence, the coding sequence being operably linked
to regulatory elements are disclosed. Methods of immunizing individuals
are disclosed.

L19 ANSWER 60 OF 178 USPATFULL
AN 2001:55710 USPATFULL
TI Materials and methods for detection and **treatment** of immune system dysfunctions
IN Clare-Salzler, Michael, Gainesville, FL, United States
PA University of Florida, Gainesville, FL, United States (U.S. corporation)
PI US 6218133 B1 20010417
AI US 1999-322628 19990528 (9)
RLI Division of Ser. No. US 1997-916586, filed on 22 Aug 1997
Continuation-in-part of Ser. No. US 1996-701928, filed on 23 Aug 1996,
now patented, Pat. No. US 5939069
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Saliwanchik, Lloyd & Saliwanchik
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 798
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The subject invention concerns novel materials and methods for the
treatment and/or prevention of autoimmune disease. In a specific embodiment, elevated production of prostaglandin synthase-2 (PGS-2) is
correlated with autoimmune dysfunction.

L19 ANSWER 61 OF 178 USPATFULL
AN 2001:55709 USPATFULL
TI Method for monitoring T cell reactivity
IN Spack, Edward G., Mountain View, CA, United States
Wehner, Nancy G., Fremont, CA, United States
McCutcheon, Michael A., Stanford, CA, United States
PA Anergen, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 6218132 B1 20010417
AI US 1997-977650 19971124 (8)
RLI Continuation-in-part of Ser. No. WO 1997-US8699, filed on 20 May 1997
Continuation-in-part of Ser. No. US 1996-657939, filed on 31 May 1996,
now patented, Pat. No. US 5750356
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Townsend and Townsend and Crew LLP
CLMN Number of Claims: 32
ECL Exemplary Claim: 1
DRWN 19 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 1770
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides a highly sensitive assay for the detection of
T-cells reactive to an antigen by detecting a soluble factor whose
secretion is induced by stimulation of the T-cell by the antigen. The
assay includes an antigen-driven proliferation of specific T cells prior
to restimulation with irradiated antigen presenting cells (APCs) and
antigen. In exemplary embodiments the assay is used to enhance the
detection limits of human peripheral blood mononuclear cells (PBMCs)
secreting interferon-.gamma. (IFN-Y) and interleukin-2 (IL-2). The assay
can be performed on previously frozen PBMCs, providing greater
convenience in sample processing, multiple use of a single sample as an
internal standard, and simultaneous analysis of samples collected at
different time points.

L19 ANSWER 62 OF 178 USPATFULL
AN 2001:55447 USPATFULL
TI Pretargeting methods and compounds
IN Meyer, Damon L., Bellevue, WA, United States
Mallett, Robert W., Seattle, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 6217869 B1 20010417
AI US 1997-926336 19970905 (8)
RLI Continuation of Ser. No. US 1994-351005, filed on 7 Dec 1994, now
abandoned Continuation-in-part of Ser. No. US 163188, now abandoned

Continuation-in-part of Ser. No. US 1992-995381, filed on 23 Dec 1992,
now abandoned Continuation-in-part of Ser. No. US 1992-895588, filed on
9 Jun 1992, now patented, Pat. No. US 5283342
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Seed Intellectual Property Law Group PLLC
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 6397
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods, compounds, compositions and kits that relate to pretargeted
delivery of diagnostic and therapeutic agents are disclosed.

L19 ANSWER 63 OF 178 USPATFULL
AN 2001:52062 USPATFULL
TI Thienodipyridine derivatives, production and use thereof
IN Sohda, Takashi, Takatsuki, Japan
Makino, Haruhiko, Hyogo, Japan
Baba, Atsuo, Ashiya, Japan
Yamane, Taihei, Ikeda, Japan
PA Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)
PI US 6214838 B1 20010410
WO 9965916 19991223
AI US 1999-355218 19990723 (9)
WO 1999-JP3155 19990614
19990723 PCT 371 date
19990723 PCT 102(e) date
PRAI JP 1998-166910 19980615
DT Utility
FS Granted
EXNAM Primary Examiner: Huang, Evelyn Mei
LREP Riesen, Philippe Y., Chao, Mark
CLMN Number of Claims: 33
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1733
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A compound of the formula (I): ##STR1##

wherein R is hydrogen or C.sub.2-6 alkanoyl; X is halogen; and ring A is
benzene ring which is optionally substituted by 1 to 4 substituents
selected from 1 halogen, 2 hydroxy, 3 C.sub.1-6 alkoxy optionally
substituted by halogen or phenyl, 4 C.sub.1-6 alkylthio optionally
substituted by halogen or phenyl, 5 C.sub.1-6 alkyl optionally
substituted by halogen, 6 C.sub.2-6 alkanoylamino or 7 carboxy
optionally esterified by C.sub.1-6 alkyl, or a salt thereof; which can
be used for preventing or treating inflammatory disease, **arthritis**, chronic rheumatoid **arthritis**, autoimmune

diseases, or rejection after organ transplantation.

L19 ANSWER 64 OF 178 USPATFULL
AN 2001:43703 USPATFULL
TI Therapeutic applications of high dose interferon
IN Tovey, Michael Gerard, Paris, France
PA Pharma Pacific Pty Ltd., New South Wales, Australia (non-U.S. corporation)
PI US 6207145 B1 20010327
AI US 1997-853870 19970509 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Goldberg, Jerome D.
LREP Browdy And Neimark
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1221
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Interferon composition for oromucosal contact to stimulate host defense mechanisms or an immune response in a mammal with a stimulating amount of the interferon which exceeds parenterally administered amounts of interferon, methods of **treatment** with such compositions and uses of interferon in the preparation of such oromucosal compositions.

L19 ANSWER 65 OF 178 USPATFULL
AN 2001:40475 USPATFULL
TI Inhibitors of interleukin-1.beta. Converting enzyme inhibitors
IN Batchelor, Mark James, Cumnor Hill, United Kingdom
Bebbington, David, Pewsey, United Kingdom
Bemis, Guy W., Arlington, MA, United States
Fridman, Wolf Herman, Paris, France
Gillespie, Roger John, Malmesbury, United Kingdom
Golec, Julian M. C., Swindon, United Kingdom
Gu, Yong, Brookline, MA, United States
Lauffer, David J., Stow, MA, United States
Livingston, David J., Newtonville, MA, United States
Matharu, Saroop Singh, Cricklade, United Kingdom
Mullican, Michael D., Needham, MA, United States
Murcko, Mark A., Holliston, MA, United States
Murdoch, Robert, Highworth, United Kingdom
Nyce, Philip, Milbury, MA, United States
Robidoux, Andrea L. C., Andover, MA, United States
Su, Michael, Newton, MA, United States
Wannamaker, M. Woods, Stow, MA, United States
Wilson, Keith P., Hopkinton, MA, United States
Zelle, Robert E., Stow, MA, United States
PA Vertex Pharmaceuticals Incorporated, Cambridge, MA, United States (U.S. corporation)
PI US 6204261 B1 20010320
AI US 1996-761483 19961206 (8)
RLI Continuation-in-part of Ser. No. US 1996-712878, filed on 12 Sep 1996
Continuation-in-part of Ser. No. US 1996-598332, filed on 8 Feb 1996,

now patented, Pat. No. US 5874424 Continuation-in-part of Ser. No. US
1995-575641, filed on 20 Dec 1995
PRAI US 1996-31495 19961126 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Gupta, Yogendra N.; Assistant Examiner: Kifle, Bruck
LREP Fish & Neave, Haley, Jr., James F., Dixon, Lisa A.
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 11 Drawing Page(s)
LN.CNT 12975
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to pyradazino[1,2-a][1,2]diazepine-1-
carboxamide compounds of formula: ##STR1##

which compounds are inhibitors of interleukin-1beta converting enzyme.

L19 ANSWER 66 OF 178 USPATFULL
AN 2001:40232 USPATFULL
TI Polynucleotide encoding a histamine receptor
IN Behan, Jiang X., Edison, NJ, United States
Hedrick, Joseph A., South River, NJ, United States
Laz, Thomas M., Parlin, NJ, United States
Monsma, Frederick J., Summit, NJ, United States
Morse, Kelley L., Livingston, NJ, United States
Umland, Shelby P., Boonton Township, NJ, United States
Wang, Suke, Edison, NJ, United States
PA Schering Corporation, Kenilworth, NJ, United States (U.S. corporation)
PI US 6204017 B1 20010320
AI US 1999-414010 19991007 (9)
DT Utility
FS Granted
EXNAM Primary Examiner: Mertz, Prema; Assistant Examiner: Murphy, Joseph F.
LREP Thampoe, Immac J.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1648
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated mammalian histamine receptor,
isolated or recombinant nucleic acids and recombinant vectors encoding
the same, host cells comprising the nucleic acids and vectors, and
methods of making the receptor using the host cells. This invention
further provides **antibodies** and antigen binding fragments
thereof which specifically bind to the receptor and are useful for
treating medical conditions caused or mediated by histamine. Also
provided are screening methods for identifying specific agonists and

antagonists of the mammalian histamine receptor.

L19 ANSWER 67 OF 178 USPATFULL
AN 2001:33252 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Carrano, Richard A., Paoli, PA, United States
Wang, Bin, Haidian, China
Weiner, David B., Merion, PA, United States
PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
Apollan, Inc., Malvern, PA, United States (U.S. corporation)
PI US 6197755 B1 20010306
AI US 1999-321461 19990527 (9)
RLI Continuation of Ser. No. US 704701, now patented, Pat. No. US 5962428
Continuation of Ser. No. US 1994-221579, filed on 1 Apr 1994, now
patented, Pat. No. US 5739118, issued on 14 Apr 1998
DT Utility
FS Granted
EXNAM Primary Examiner: Schwartzman, Robert A.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 3329
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of introducing genetic material into cells of an individual and
compositions and kits for practicing the same are disclosed. The methods
comprise the steps of contacting cells of an individual with a genetic
vaccine facilitator and administering to the cells, a nucleic acid
molecule that is free of retroviral particles. The nucleic acid molecule
comprises a nucleotide sequence that encodes a protein that comprises at
least one epitope that is identical or substantially similar to an
epitope of a pathogen antigen or an antigen associated with a
hyperproliferative or autoimmune disease, a protein otherwise missing
from the individual due to a missing, non-functional or partially
functioning gene, or a protein that produce a therapeutic effect on an
individual. Methods of prophylactically and therapeutically immunizing
an individual against HIV are disclosed. Pharmaceutical compositions and
kits for practicing methods of the present invention are disclosed.

L19 ANSWER 68 OF 178 USPATFULL
AN 2001:33021 USPATFULL
TI Methods for detecting, identifying, isolating, and selectively labelling

and targeting TH1 lymphocyte by means of the LAG-3 protein
IN Romagnani, Sergio, Florence, Italy
PA Institute National de la Sante et de la Recherche Medicale, Paris,
France (non-U.S. corporation)
Institut Gustave Roussy, Villejuif Cedex, France (non-U.S. corporation)
Applied Research Systems, ARS Holding N.V., Curacao, Netherlands
Antilles (non-U.S. corporation)
PI US 6197524 B1 20010306
WO 9703695 19970206
AI US 1998-983576 19980415 (8)
WO 1996-US11994 19960719
19980415 PCT 371 date
19980415 PCT 102(e) date
PRAI US 1995-1367 19950721 (60)
US 1995-2683 19950921 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Schwadron, Ronald B.
LREP Browdy and Neimark
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1033
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The lymphocyte ativation gene (LAG-3) is a member of the immunoglobulin
superfamily that is selectively transcribed in human activated T and NK
cells. Surface LAG-3 expression correlated with IFN-.gamma. but not
IL-4, production in antigen-stimulated T-cells and it was up-regulated
by **IL-12** and is preferentially associated with CD4+
T-cells producing Th1-type cytokines. The presence of LAG-3 on the
surface of Th1 lymphocytes is used as a marker to detect and identify
Th1 lymphocytes and differentiate them from Th2 lymphocytes. Monoclonal
antibodies to LAG-3 are used in methods of detecting and
isolating Th1 cells as well as methods of diagnosing Th1-mediated
disease. The present invention also relates to methods of treating
infectious diseases, cancer, and disorders assocated with Th1/Th2
imbalance.

L19 ANSWER 69 OF 178 USPATFULL
AN 2001:29605 USPATFULL
TI Protein kinase inhibitor
IN Sriram, Subramaniam, Nashville, TN, United States
Bright, John, Nashville, TN, United States
Nag, Bishwajit, Fremont, CA, United States
Sharma, Somesh D., Los Altos, CA, United States
PA Calyx Therapeutics, Inc., Hayward, CA, United States (U.S. corporation)

PI US 6194453 B1 20010227
AI US 1998-218264 19981221 (9)
RLI Continuation of Ser. No. US 1997-825662, filed on 3 Apr 1997, now
patented, Pat. No. US 5854285, issued on 29 Dec 1998
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Fish & Richardson, P.C.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 276
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed herein are compounds of the formula I ##STR1##

wherein A and C are independently H, alkyl of 1-6 carbon atoms, hydroxy,
or alkoxy of 1-6 carbon atoms;

B is hydroxy or alkoxy of 1-6 carbon atoms; and

Y is cyano, ##STR2##

--C(NR.sub.1 R.sub.2).dbd.C(CN).sub.2 ;

wherein X.dbd.O or S, and R.sub.1 and R.sub.2 are independently

H, benzyl, --CH(CH.sub.3)C.sub.6 H.sub.6,

--(CH.sub.2).sub.n C.sub.6 H.sub.6, phenyl; --CO.sub.2 R;

n=2-4; R is lower alkyl of 1-6 carbon atoms which are useful for
treating inflammation and immunological diseases.

L19 ANSWER 70 OF 178 USPATFULL
AN 2001:29360 USPATFULL
TI Methods for the selective expansion of lymphocytes by in vitro cultivation
IN Bell, David N., Oakville, Canada
Wong, Truman, North York, Canada
PA Hemosol Inc., Etobicoke, Canada (non-U.S. corporation)
PI US 6194207 B1 20010227
AI US 1998-16784 19980130 (9)
PRAI US 1997-37245 19970131 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: Tung, Mary Beth
LREP Bereskin & Parr, Gravelle, Micheline
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 29 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 1248
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to methods for the production of selected
populations of lymphocytes. Lymphocytes produced can be isolated and

purified using well known and established procedures to provide a consistent lymphocyte source which one of ordinary skill in the art can modify to provide an appropriate type or an optimal level of a desired lymphocyte. The availability of such cell populations allows for not only for the complete reconstitution of the depleted, defective or missing lymphocyte population in a patient, but also provides the flexibility of having sufficient cells to permit multiple or cyclic treatments. These methods for expanding target cell populations are broadly applicable to the selective expansion of several types of lymphocytes and are demonstrated to maintain phenotype as well as antigen specificity.

L19 ANSWER 71 OF 178 USPATFULL
AN 2001:29120 USPATFULL
TI Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor **antibodies**
IN Deo, Yashwant M., Audubon, PA, United States
Graziano, Robert, Frenchtown, NJ, United States
Keler, Tibor, Ottsville, PA, United States
PA Mederax, Inc., Annandale, NJ, United States (U.S. corporation)
PI US 6193966 B1 20010227
AI US 1997-890011 19970710 (8)
RLI Continuation-in-part of Ser. No. US 1996-678194, filed on 11 Jul 1996, now patented, Pat. No. US 5922845
DT Utility
FS Granted
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Lahive & Cockfield, LLP, Remillard, Esq., Jane E.
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 30 Drawing Figure(s); 28 Drawing Page(s)
LN.CNT 2686
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor **antibodies** and methods of use are provided.

L19 ANSWER 72 OF 178 USPATFULL
AN 2001:18199 USPATFULL
TI Methods of diagnosing clinical subtypes of crohn's disease with characteristic responsiveness to anti-Th1 cytokine **therapy**
IN Plevy, Scott E., Tenafly, NJ, United States
Targan, Stephan R., Santa Monica, CA, United States
Taylor, Kent, Santa Paula, CA, United States
Barry, Mary J., Ramona, CA, United States
PA Prometheus Laboratories, Inc., San Diego, CA, United States (U.S.

corporation)
PI US 6183951 B1 20010206
AI US 1997-855825 19970512 (8)
RLI Continuation-in-part of Ser. No. US 1997-837056, filed on 11 Apr 1997,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Lazar-Wesley,
Eliane
LREP Campbell & Flores, LLP
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2561
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods based on serological and genetic
markers for diagnosing clinical subtypes of Crohn's disease (CD) having
characteristic responsiveness to anti-Th1 cytokine **therapy**. In the methods of the inventions the presence of perinuclear anti-neutrophil **antibody** (pANCA), the presence of the TNFa10b4c1d3e3 haplotype or the presence TNFa11b4c1d3e3 haplotype each
are independently diagnostic of a clinical subtype of CD having an
inferior clinical response to anti-Th1 cytokine **therapy**. In addition, the presence of the homozygous **TNF**-.beta. 1111 haplotype involving the TNFc, aa13L, aa26 and NcoI loci is independently
diagnostic of a clinical subtype of CD having an inferior clinical
response to anti-Th1 cytokine **therapy**. The presence of speckling anti-pan polymorphonuclear **antibody** (SAPPA) is diagnostic of a clinical subtype of CD having a superior clinical
response to anti-Th1 cytokine **therapy**.

L19 ANSWER 73 OF 178 USPATFULL
AN 2001:7889 USPATFULL
TI In-vitro transcription processes for screening natural products and
other chemical substances
IN Kirschbaum, Bernd, Mainz, Germany, Federal Republic of
Stahl, Wilhelm, Idstein, Germany, Federal Republic of
Winkler, Irvin, Liederbach, Germany, Federal Republic of
Meisterernst, Michael, Eichenau, Germany, Federal Republic of
PA Aventis Pharma Deutschland GmbH, Germany, Federal Republic of (non-U.S.
corporation)
PI US 6174722 B1 20010116
AI US 1998-38141 19980311 (9)
PRAI DE 1997-19710159 19970312
DT Utility
FS Granted
EXNAM Primary Examiner: Horlick, Kenneth R.; Assistant Examiner: Siew, Jeffrey

LREP Foley & Lardner
CLMN Number of Claims: 8
ECL Exemplary Claim: 5
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1418
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An process for analyzing transcription which can be automated and which
is suitable for bulk screening. The process involves transcribing a DNA
sequence using a nuclear extract, which can be complemented or fully
replaced by exogenous transcription factors and/or cofactors; optionally
removing the proteins of the reaction mixture; binding the resulting
transcript to a solid matrix; removing the excess labeled nucleotides;
and determining the amount of labeled transcript. Methods of using the
inventive process to identify compounds having a selective effect on
gene expression.

L19 ANSWER 74 OF 178 USPATFULL
AN 2001:1480 USPATFULL
TI Materials and methods for detection and **treatment** of immune system dysfunctions
IN Clare-Salzler, Michael, Gainesville, FL, United States
PA University of Florida, Gainesville, FL, United States (U.S. corporation)
PI US 6168792 B1 20010102
AI US 1997-916586 19970822 (8)
RLI Continuation-in-part of Ser. No. US 1996-701928, filed on 23 Aug 1996,
now patented, Pat. No. US 5939069
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Saliwanchik, Lloyd & Saliwanchik
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 859
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The subject invention concerns novel materials and methods for the
treatment and/or prevention of autoimmune disease. In a specific
embodiment, elevated production of prostaglandin synthase-2 (PGS-2) is
correlated with autoimmune dysfunction.

L19 ANSWER 75 OF 178 USPATFULL
AN 2000:174812 USPATFULL
TI Monoclonal **antibody** which binds to a human-Th2-specific protein and hybridoma
IN Ogawa, Kazuyuki, Saitama, Japan
Tanaka, Kazuya, Saitama, Japan
Nagata, Kinya, Saitama, Japan

Takano, Syoichi, Saitama, Japan
PA BML, Inc., Japan (non-U.S. corporation)
PI US 6166186 20001226
AI US 2000-480784 20000110 (9)
RLI Division of Ser. No. US 981825
PRAI JP 1996-166793 19960605
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: DeCloux, Amy
LREP Knobbe Martens Olson & Bear, LLP
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1273
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present specification provides a means for specifying the condition and type of immune-related diseases on the basis of knowledge about the polarization of the distribution of helper T-cell subsets Th1 and Th2.

More specifically, the gene (B19), specific only to the human Th2, is prepared by a subtraction method. A human-Th2-specific protein which the gene encodes is produced by recombinant methods, and a monoclonal **antibody** against the Th2-specific protein and a hybridoma which produces the monoclonal **antibody** is provided.

L19 ANSWER 76 OF 178 USPATFULL
AN 2000:168140 USPATFULL
TI Compounds and methods for **treatment** and diagnosis of mycobacterial infections
IN Visser, Elizabeth, Auckland, New Zealand
PA Genesis Researth and Development Corporation Limited, Parnell, New Zealand (non-U.S. corporation)
PI US 6160093 20001212
AI US 1998-95855 19980611 (9)
RLI Continuation-in-part of Ser. No. US 1997-997362, filed on 23 Dec 1997, now patented, Pat. No. US 5985287 which is a continuation-in-part of Ser. No. US 1997-873970, filed on 12 Jun 1997, now patented, Pat. No. US 6001361 which is a continuation-in-part of Ser. No. US 1996-705347, filed on 29 Aug 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Mosher, Mary E.
LREP Speckman, Ann W., Sleath, Janet
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 7369
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides polypeptides comprising an immunogenic portion of a M. vaccae protein and DNA molecules encoding such polypeptides, together with methods for their use in the diagnosis and **treatment** of mycobacterial infection. Methods for enhancing the immune response to an antigen including administration of M. vaccae culture filtrate, delipidated M. vaccae cells or delipidated and deglycolipidated M. vaccae cells are also provided.

L19 ANSWER 77 OF 178 USPATFULL
AN 2000:157632 USPATFULL
TI Human **antibodies** derived from immunized xenomice
IN Kucherlapati, Raju, Darien, CT, United States
Jakobovits, Aya, Menlo Park, CA, United States
Brenner, Daniel G., Redwood City, CA, United States
Capon, Daniel J., Hillsborough, CA, United States
Klapholz, Sue, Stanford, CA, United States
PA Abgenix, Inc., Fremont, CA, United States (U.S. corporation)
PI US 6150584 20001121
AI US 1996-724752 19961002 (8)
RLI Continuation-in-part of Ser. No. US 1995-430938, filed on 27 Apr 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-234143, filed on 28 Apr 1994, now abandoned And a continuation-in-part of Ser. No. US 1993-112848, filed on 27 Aug 1993, now abandoned And a continuation-in-part of Ser. No. US 1993-31801, filed on 15 Mar 1993 And a continuation-in-part of Ser. No. US 1992-919297, filed on 24 Jul 1992, now abandoned And a continuation-in-part of Ser. No. US 1990-610515, filed on 8 Nov 1990, now abandoned And a continuation-in-part of Ser. No. US 1990-466008, filed on 12 Jan 1990, now abandoned And a continuation-in-part of Ser. No. WO 1996-US5928, filed on 29 Apr 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Hauda, Karen M.
LREP Fish & Neave, Haley, Jr., James F., Gunnison, Jane T.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 26 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 1872
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Fully human **antibodies** against a specific antigen can be prepared by administering the antigen to a transgenic animal which has been modified to produce such **antibodies** in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either **antibodies** per se or analogs thereof.

L19 ANSWER 78 OF 178 USPATFULL
AN 2000:153471 USPATFULL
TI Cyanidin compositions and therapeutic and diagnostic uses therefor
IN van de Winkel, Jan G. J., Odijk, Netherlands
PA Medarex, Inc., Annandale, NJ, United States (U.S. corporation)
PI US 6146837 20001114
AI US 1998-197683 19981123 (9)
RLI Division of Ser. No. US 1996-709411, filed on 6 Sep 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Lubet, Martha
LREP Lahive & Cockfield, LLPJane E. Remillard, EsquireJeanne M. DiGiorgio, Esq.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1340
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions comprising cyanidin reagents for binding to Fc.gamma.RI receptors, and methods and kits for use therefor are provided.

L19 ANSWER 79 OF 178 USPATFULL
AN 2000:138395 USPATFULL
TI **Treatment** of T-helper cell type 2-mediated immune disease by retinoid **antagonists**
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Panina-Bordignon, Paola, Milan, Italy
Sinigaglia, Francesco, Milan, Italy
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6133309 20001017
AI US 1998-189189 19981110 (9)
PRAI EP 1997-119776 19971112
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Johnston, George W., Epstein, William H., Parise, John P.
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Retinoids with retinoid receptor **antagonistic** activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L19 ANSWER 80 OF 178 USPATFULL
AN 2000:131642 USPATFULL
TI Multifunctional complexes for gene transfer into cells comprising a

nucleic acid bound to a polyamine and having a endosome disruption agent
IN Boutin, Raymond H., Thornton, PA, United States
PA American Home Products Corporation, Madison, NJ, United States (U.S. corporation)
PI US 6127170 20001003
WO 9610038 19960404
AI US 1997-809397 19970321 (8)
WO 1995-US12502 19950928
19970321 PCT 371 date
19970321 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1994-314060, filed on 28 Sep 1994, now patented, Pat. No. US 5837533, issued on 17 Nov 1998
DT Utility
FS Granted
EXNAM Primary Examiner: Crouch, Deborah
LREP Howson and Howson
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 4293
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A multifunctional molecular complex for the transfer of a nucleic acid composition to a target cell is provided. The complex is comprised of A) said nucleic acid composition and B) a transfer moiety comprising 1) one or more cationic polyamines bound to said nucleic acid composition, 2) one or more endosome membrane disrupting components attached to at least one nitrogen of the polyamine and 3) one or more receptor specific binding components.

L19 ANSWER 81 OF 178 USPATFULL
AN 2000:131407 USPATFULL
TI Methods of treating inflammatory bowel diseases by administering IL-11
IN Warne, Nick W., Andover, MA, United States
Bedrosian, Camille L., Belmont Hills, MA, United States
Keith, Jr., James C., Andover, MA, United States
Schwertschlag, Ullrich S., Beverly Farms, MA, United States
Schendel, Paul F., Wayland, MA, United States
PA Genetics Institute, Cambridge, MA, United States (U.S. corporation)
PI US 6126933 20001003
AI US 1998-179026 19981026 (9)
RLI Continuation-in-part of Ser. No. US 1997-892407, filed on 15 Jul 1997, now patented, Pat. No. US 5948402 which is a division of Ser. No. US 1995-495724, filed on 27 Jun 1995, now patented, Pat. No. US 5679339, issued on 21 Oct 1997
PRAI WO 1996-US8059 19960530

DT Utility
FS Granted
EXNAM Primary Examiner: Mertz, Prema
LREP Cserr, Luann, Gyure, Barbara
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1036
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Provided by the present invention are topical formulations of Interleukin-11 and methods for treating a variety of disorders, including inflammatory bowel diseases (e.g., Crohn's disease, ulcerative colitis, indeterminate colitis, and infectious colitis), mucositis (e.g., oral mucositis, gastrointestinal mucositis, nasal mucositis, and proctitis), necrotizing enterocolitis, inflammatory skin disorders (e.g., psoriasis, atopic dermatitis, and contact hypersensitivity), aphthous ulcers, pharyngitis, esophagitis, peptic ulcers, gingivitis, periodontitis, and ocular diseases (e.g., conjunctivitis, retinitis, and uveitis).

L19 ANSWER 82 OF 178 USPATFULL
AN 2000:117526 USPATFULL
TI Synferon, a synthetic interferon
IN Olsen, Henrik S., Gaithersburg, MD, United States
Gentz, Reiner L., Rockville, MD, United States
Ruben, Steven M., Olney, MD, United States
PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)
PI US 6114145 20000905
AI US 1998-205264 19981202 (9)
PRAI US 1997-67746 19971205 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Fitzgerald, David L.
LREP Human Genome Sciences Inc.
CLMN Number of Claims: 149
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2474
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a novel Synferon protein which is a member of the interferon family. In particular, isolated nucleic acid molecules are provided encoding a synthetic interferon polypeptide, called "Synferon". Synferon polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists

and **antagonists** of Synferon activity. Also provided are therapeutic methods for treating immune system-related disorders.

L19 ANSWER 83 OF 178 USPATFULL
AN 2000:98398 USPATFULL
TI Pharmaceutical angiostatic dipeptide compositions and methods of use thereof
IN Green, Lawrence R., Tacoma, WA, United States
Blasecki, John W., Woodinville, WA, United States
PA Cytran, Inc., Kirkland, WA, United States (U.S. corporation)
PI US 6096713 20000801
AI US 1999-260190 19990301 (9)
RLI Continuation of Ser. No. US 1996-614764, filed on 13 Mar 1996, now patented, Pat. No. US 5902790 which is a continuation-in-part of Ser. No. US 1995-538701, filed on 3 Oct 1995, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Celsa, Bennett
LREP Townsend and Townsend and Crew
CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1150
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods of inhibiting neovascularization in a subject by administering to the subject a pharmaceutical preparation of R'-Glu-Trp-R".

L19 ANSWER 84 OF 178 USPATFULL
AN 2000:95042 USPATFULL
TI Therapeutic methods employing disulfide derivatives of dithiocarbamates and compositions useful therefor
IN Lai, Ching-San, Encinitas, CA, United States
Vassilev, Vassil, San Diego, CA, United States
PA Medinox Inc., San Diego, CA, United States (U.S. corporation)
PI US 6093743 20000725
AI US 1998-103639 19980623 (9)
DT Utility
FS Granted
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Gary Cary Ware & Freidenrich, Reiter, Stephen E., Kirschenbaum, Shelia R.
CLMN Number of Claims: 51
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2691
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a novel dithiocarbamate disulfide dimer useful in various therapeutic treatments, either alone or in combination with other active agents. In one method, the disulfide derivative of a

dithiocarbamate is coadministered with an agent that inactivates (or inhibits the production of) species that induce the expression of nitric oxide synthase to reduce the production of such species, while, at the same time reducing nitric oxide levels in the subject. In another embodiment, free iron ion levels are reduced in a subject by administration of a disulfide derivative of a dithiocarbamate(s) to scavenge free iron ions, for example, in subjects undergoing anthracycline chemotherapy. In another embodiment, cyanide levels are reduced in a subject by administration of a disulfide derivative of a dithiocarbamate so as to bind cyanide in the subject. In a further aspect, the present invention relates to compositions and formulations useful in such therapeutic methods.

L19 ANSWER 85 OF 178 USPATFULL
AN 2000:87707 USPATFULL
TI Methods and compositions for the inhibition of interleukin-12 production
IN Karp, Christopher L., Lutherville, MD, United States
Trinchieri, Giorgio, Wynnewood, PA, United States
Wysocka, Maria, Wynnewood, PA, United States
Griffin, Diane E., Hunt Valley, MD, United States
PA The Wistar Insitute, Philadelphia, PA, United States (U.S. corporation)
Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)
PI US 6086876 20000711
AI US 1998-19862 19980206 (9)
PRAI US 1997-37722 19970207 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Kemmerer, Elizabeth; Assistant Examiner: Romeo, David S.
LREP Akin, Gump, Strauss, Hauer & Feld, L.L.P.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 1487
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention includes compositions and methods for selective suppression of **IL-12** production in a cell. Methods of treating a human having a disease associated with dysregulated **IL-12** production are also provided.

L19 ANSWER 86 OF 178 USPATFULL
AN 2000:80771 USPATFULL
TI Aromatic heterocyclic compounds and their use as anti-inflammatory agents

IN Regan, John R., Larchmont, NY, United States
Cirillo, Pier F., Woodbury, CT, United States
Hickey, Eugene R., Danbury, CT, United States
Moss, Neil, Ridgefield, CT, United States
Cywin, Charles L., Bethel, CT, United States
Pargellis, Christopher, West Redding, CT, United States
Gilmore, Thomas A., Middlebury, CT, United States
PA Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT, United
States (U.S. corporation)
PI US 6080763 20000627
AI US 1998-181743 19981029 (9)
PRAI US 1997-64102 19971103 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Owens, Amelia
LREP Raymond, Robert P., Bottino, Anthony P., Stempel, Alan R.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2027
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel aromatic heterocyclic compounds inhibit cytokines production
involved in immunoregulation and inflammation such as interleukin-1 and
tumor necrosis factor production. The compounds are therefore useful in
pharmaceutic compositions for treating diseases or pathological
conditions involving inflammation such as chronic inflammatory diseases.

L19 ANSWER 87 OF 178 USPATFULL
AN 2000:80750 USPATFULL
TI Substituted benzamides
IN Germann, Tieno, Herzogenrath, Germany, Federal Republic of
Frosch, Stefanie, Aachen, Germany, Federal Republic of
Zimmer, Oswald, Wuerselen, Germany, Federal Republic of
PA Gruenenthal GmbH, Aachen, Germany, Federal Republic of (non-U.S.
corporation)
PI US 6080742 20000627
AI US 1999-405180 19990924 (9)
PRAI DE 1998-19843793 19980924
DT Utility
FS Granted
EXNAM Primary Examiner: Stockton, Laura L.
LREP Evenson, McKeown, Edwards & Lenahan, P.L.L.C.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 426
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Substituted benzamides corresponding to the formula I ##STR1## wherein
R.sup.1, R.sup.2 and R.sup.3 have the meanings given herein, and their
use in pharmaceutical compositions. The compounds are particularly

useful as immunomodulators.

L19 ANSWER 88 OF 178 USPATFULL
AN 2000:74445 USPATFULL
TI Human **antibodies** derived from immunized xenomice
IN Kucherlapati, Raju, Darien, CT, United States
Jakobovits, Aya, Menlo Park, CA, United States
Klapholz, Sue, Stanford, CA, United States
Brenner, Daniel G., San Mateo, CA, United States
Capon, Daniel J., Hillsborough, CA, United States
PA Abgenix, Inc., Fremont, CA, United States (U.S. corporation)
PI US 6075181 20000613
AI US 1995-486857 19950607 (8)
RLI Division of Ser. No. US 1995-430938, filed on 27 Apr 1995, now abandoned
which is a continuation-in-part of Ser. No. US 1994-234145, filed on 28
Apr 1994, now abandoned which is a continuation-in-part of Ser. No. US
1993-112848, filed on 27 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-919297, filed on 24 Jul 1992,
now abandoned which is a continuation-in-part of Ser. No. US 1990-610515, filed on 8 Nov 1990, now abandoned which is a continuation-in-part of Ser. No. US 1990-466008, filed on 12 Jan 1990,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Hauda, Karen M.
LREP Fish & Neave, Haley, Jr., James F., Gunnison, Jane T.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 17 Drawing Page(s)
LN.CNT 1233
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB **Antibodies** with fully human variable regions against a specific antigen can be prepared by administering the antigen to a
transgenic animal which has been modified to produce such **antibodies** in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can
be performed to obtain either **antibodies** per se or analogs thereof.

L19 ANSWER 89 OF 178 USPATFULL
AN 2000:74115 USPATFULL
TI Polynucleotides encoding human CTLA-8 related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S.

corporation)
PI US 6074849 20000613
AI US 1996-685239 19960718 (8)
RLI Continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1658
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 related proteins are disclosed.

Human CTLA-8 proteins and methods for their production are also disclosed. Methods of **treatment** using human CTLA-8 proteins, rat CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L19 ANSWER 90 OF 178 USPATFULL
AN 2000:70826 USPATFULL
TI Use of vitamin D compounds to prevent transplant rejection
IN DeLuca, Hector F., Deerfield, WI, United States
Cantorna, Margherita T., Middleton, WI, United States
Hayes, Colleen E., Madison, WI, United States
Hullett, Debra A., Madison, WI, United States
Sollinger, Hans W., Madison, WI, United States
Humpal-Winter, Jean, Madison, WI, United States
PA Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)
PI US 6071897 20000606
AI US 1998-115958 19980715 (9)
RLI Continuation-in-part of Ser. No. US 1997-870569, filed on 6 Jun 1997, now abandoned And a continuation-in-part of Ser. No. US 1997-870337, filed on 6 Jun 1997, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Quarles & Brady LLP
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 827
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of moderating transplant rejection in a transplant recipient comprising administering a dose of vitamin D compound effective to prevent transplant rejection is disclosed. Preferably, the recipient's susceptibility to opportunistic infections has not been compromised.

Also preferably, the recipient has not suffered bone demineralization.

L19 ANSWER 91 OF 178 USPATFULL
AN 2000:50737 USPATFULL
TI Methods and compositions for modulating responsiveness to corticosteroids
IN Sekut, Les, Westborough, MA, United States
Carter, Adam, Newburyport, MA, United States
Ghayur, Tariq, Grafton, MA, United States
Banerjee, Subhashis, Shrewsbury, MA, United States
Tracey, Daniel E., Harvard, MA, United States
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of
(non-U.S. corporation)
PI US 6054487 20000425
AI US 1997-820692 19970318 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Lahive & Cockfield, LLP
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.
In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) **antagonist**. In another embodiment, the agent is an interleukin-12 (**IL-12**) **antagonist**. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment, the agent is an anti-**IL-12** monoclonal **antibody**. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the **treatment** of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable

carrier.

L19 ANSWER 92 OF 178 USPATFULL
AN 2000:41155 USPATFULL
TI FAS ligand fusion proteins and their uses
IN Queen, Cary L., Los Altos, CA, United States
Schneider, William P., Los Altos, CA, United States
Vasquez, Maximiliano, Palo Alto, CA, United States
PA Protein Design Labs., Inc., Fremont, CA, United States (U.S. corporation)
PI US 6046310 20000404
AI US 1997-815190 19970311 (8)
RLI Continuation-in-part of Ser. No. US 1996-614584, filed on 13 Mar 1996,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Nolan, Patrick
J.
LREP Townsend & Townsend & Crew LLP
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1454
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Fas ligand fusion proteins comprising a polypeptide capable of specifically binding an antigen or a cell surface marker are prepared
employing recombinant DNA technology for use in, e.g., **treatment**
of autoimmune disorders.

L19 ANSWER 93 OF 178 USPATFULL
AN 2000:40880 USPATFULL
TI Polynucleotides encoding a cardiotrophin-like cytokine
IN Shi, Yanggu, Gaithersburg, MD, United States
Ruben, Steven M., Olney, MD, United States
PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)
PI US 6046035 20000404
AI US 1998-106182 19980629 (9)
PRAI US 1997-51311 19970630 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John; Assistant Examiner: Saoud, Christine
LREP Human Genome Sciences Inc.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 3830
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a novel CLC protein which is a member
of the IL-6 cytokine family. In particular, isolated nucleic acid
molecules are provided encoding the human CLC protein. CLC polypeptides

are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and **antagonists** of CLC activity. Also provided are diagnostic methods for detecting cardiac and immune system-related disorders and therapeutic methods for treating cardiac and immune system-related disorders.

L19 ANSWER 94 OF 178 USPATFULL
AN 2000:37900 USPATFULL
TI Human CTLA-8 and uses of CTLA-8-related proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
Goldman, Samuel, Acton, MA, United States
Pittman, Debra, Windham, NH, United States
Mi, Sha, Belmont, MA, United States
Neben, Steven, Acton, MA, United States
Giannotti, Joanne, Acton, MA, United States
Golden-Fleet, Margaret M., Medford, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6043344 20000328
AI US 1998-34810 19980304 (9)
RLI Division of Ser. No. US 1996-685239, filed on 18 Jul 1996, now abandoned which is a continuation-in-part of Ser. No. US 1995-504032, filed on 19 Jul 1995 which is a continuation-in-part of Ser. No. US 1995-514014, filed on 11 Aug 1995, now patented, Pat. No. US 5707829
PRAI US 1995-35347 19950719 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Lahive & Cockfield, LLP, Mandragouras, Esq., Amy E., Lauro, Esq., Peter C.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1761
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polynucleotides encoding human CTLA-8 and related proteins are disclosed. Human CTLA-8 proteins and methods for their production are also disclosed. Methods of **treatment** using human CTLA-8 proteins, rat CTLA-8 proteins and herpesvirus herpes CTLA-8 proteins are also provided.

L19 ANSWER 95 OF 178 USPATFULL
AN 2000:34670 USPATFULL
TI Human Th2 specific protein
IN Ogawa, Kazuyuki, Saitama, Japan
Tanaka, Kazuya, Saitama, Japan

Nagata, Kinya, Saitama, Japan
Takano, Syoichi, Saitama, Japan
PA BML, Inc., Tokyo, Japan (non-U.S. corporation)
PI US 6040426 20000321
WO 9746677 19971211
AI US 1998-981825 19980511 (8)
WO 1997-JP1906 19970605
19980511 PCT 371 date
19980511 PCT 102(e) date
PRAI JP 1996-166793 19960605
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DeCloux, Amy
LREP Knobbe, Martens Olson & Bear, LLP
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1190
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides for specifying the condition and type of immune-related diseases on the basis of the knowledge about the polarization of the distribution of helper T-cell subsets Th1 and Th2.

More sepcifically, in this invention, the gene (B19) specific only the human Th2 is prepared and specified by a subtraction method, and a recombinant vector into which the gene is incorporated, a transformant transformed by the recombinant vector, a human-Th2-specific protein which the gene encodes and which derives from the transformant, and a monoclonal **antibody** against the Th2-specific protein are produced and the gene, protein, **antibody**, etc. are used as the means for specifying or correcting the polarization of the distribution of Th1 and Th2 to solve the above object.

L19 ANSWER 96 OF 178 USPATFULL
AN 2000:24287 USPATFULL
TI Receptor specific transepithelial transport of therapeutics
IN Blumberg, Richard S., Chestnut Hill, MA, United States
Simister, Neil E., Wellesley, MA, United States
Lencer, Wayne I., Jamaica Plain, MA, United States
PA The Brigham and Women's Hospital, Inc., Boston, MA, United States (U.S. corporation)
Brandeis University, Waltham, MA, United States (U.S. corporation)
PI US 6030613 20000229
AI US 1997-899856 19970724 (8)
RLI Continuation-in-part of Ser. No. US 1995-578171, filed on 29 Dec 1995 which is a continuation-in-part of Ser. No. US 1995-374159, filed on 17 Jan 1995, now patented, Pat. No. US 5671273

DT Utility
FS Granted
EXNAM Primary Examiner: Cunningham, Thomas M.
LREP Wolf, Greenfield & Sacks, P.C.
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1591
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates in general to methods and products for initiating an immune response against an antigen, and in particular relates to transepithelial delivery of antigens to provoke tolerance and immunity. The present invention further relates to methods and products for the transepithelial delivery of therapeutics. In particular, the invention relates to methods and compositions for the delivery of therapeutics conjugated to a FcRn binding partner to intestinal epithelium, mucosal epithelium and epithelium of the lung. The present invention further relates to the synthesis, preparation and use of the FcRn binding partner conjugates as, or in, pharmaceutical compositions for oral systemic delivery of drugs and vaccines.

L19 ANSWER 97 OF 178 USPATFULL
AN 2000:21680 USPATFULL
TI High affinity nucleic acid ligands of cytokines
IN Tasset, Diane, Boulder, CO, United States
Pagratis, Nikos, Boulder, CO, United States
Jayasena, Sumedha, Boulder, CO, United States
Gold, Larry, Boulder, CO, United States
PA NeXstar Pharmaceuticals, Inc., Boulder, CO, United States (U.S. corporation)
PI US 6028186 20000222
AI US 1995-481710 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1991-714131, filed on 10 Jun 1991, now patented, Pat. No. US 5475096 And a continuation-in-part of Ser. No. US 1992-931473, filed on 17 Aug 1992, now patented, Pat. No. US 5270163 And a continuation-in-part of Ser. No. US 1992-964624, filed on 21 Oct 1992, now patented, Pat. No. US 5496938 And a continuation-in-part of Ser. No. US 1993-117991, filed on 8 Sep 1993, now abandoned , said Ser. No. US 714131 which is a continuation-in-part of Ser. No. US 1990-536428, filed on 11 Jun 1990, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Zitomer, Stephanie
LREP Swanson & Bratschun LLC

CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 5603
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are described for the identification and preparation of high-affinity nucleic acid ligands to cytokines. Included in the invention are specific nucleic acid ligands to IFN-gamma, IL-4, IL-10, **TNF**-alpha, and RANTES.

L19 ANSWER 98 OF 178 USPATFULL
AN 2000:10020 USPATFULL
TI Binding agents specific for IgA receptor
IN Shen, Lilian, Thetford Center, VT, United States
Fanger, Michael W., Lebanon, NH, United States
PA Trustees of Dartmouth College, Hanover, NH, United States (U.S. corporation)
PI US 6018031 20000125
AI US 1996-756142 19961126 (8)
RLI Continuation-in-part of Ser. No. US 1994-222572, filed on 4 Apr 1994, now patented, Pat. No. US 5610057, issued on 11 Mar 1997 which is a continuation of Ser. No. US 1992-871561, filed on 16 Apr 1992, now abandoned which is a continuation of Ser. No. US 1989-424883, filed on 20 Oct 1989, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Ungar, Susan
LREP Lahive & Cockfield, LLP, Remillard, Jane E., DeConti, Jr, Giulio A.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1971
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Binding agents which bind specifically to a receptor for human IgA, including monoclonal **antibodies** which react specifically to Fc receptor for IgA of human effector cells are disclosed. The binding agents, e.g., **antibodies** are useful for targeting human effector cells (e.g. macrophages) against a target cell (e.g. a cancer cell, an infectious agent, etc.). For this purpose, bifunctional **antibodies** or heteroantibodies can be constructed containing the binding region derived from an anti-Fc-alpha receptor **antibody** and the binding region of a target-specific **antibody**. Targeted effector cells can specifically lyse target cells.

L19 ANSWER 99 OF 178 USPATFULL
AN 2000:7398 USPATFULL
TI Biotinamido-n-methylglycyl-seryl-o-succinamido-benzyl dota

IN Theodore, Louis J., Lynnwood, WA, United States
Kasina, Sudhakar, Kirkland, WA, United States
Reno, John M., Brier, WA, United States
Gustavson, Linda M., Seattle, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 6015897 20000118
AI US 1996-645211 19960513 (8)
RLI Division of Ser. No. US 1994-351005, filed on 7 Dec 1994, now abandoned
which is a continuation-in-part of Ser. No. US 1993-163188, filed on 7
Dec 1993, now abandoned which is a continuation-in-part of Ser. No. WO
1993-US5406, filed on 7 Jun 1993 which is a continuation-in-part of Ser.
No. US 1992-995381, filed on 23 Dec 1992, now abandoned which is a
continuation-in-part of Ser. No. US 1992-895588, filed on 9 Jun 1992,
now patented, Pat. No. US 5283342
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Gambel,
Phillip
LREP Seed and Berry LLP
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 6303
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods, compounds, compositions and kits that relate to pretargeted
delivery of diagnostic and therapeutic agents are disclosed.
Biotinamido-N-methylglycyl-seryl-O-succinamido-benzyl DOTA is disclosed.

L19 ANSWER 100 OF 178 USPATFULL
AN 2000:7385 USPATFULL
TI Soluble divalent and multivalent heterodimeric analogs of proteins
IN Schneck, Jonathan, Silver Spring, MD, United States
O'Herrin, Sean, Baltimore, MD, United States
PA The Johns Hopkins University, Baltimore, MD, United States (U.S.
corporation)
PI US 6015884 20000118
AI US 1997-828712 19970328 (8)
PRAI US 1996-14367 19960328 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Bansal, Geetha
P.
LREP Banner & Witcoff, Ltd.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 16 Drawing Page(s)

LN.CNT 2027
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Specificity in immune responses is in part controlled by the selective interaction of T cell receptors with their cognate ligands, peptide/MHC molecules. The discriminating nature of this interaction makes these molecules, in soluble form, good candidates for selectively regulating immune responses. Attempts to exploit soluble analogs of these proteins has been hampered by the intrinsic low avidity of these molecules for their ligands. To increase the avidity of soluble analogs for their cognates to biologically relevant levels, divalent peptide/MHC complexes or T cell receptors (superdimers) were constructed. Using a recombinant DNA strategy, DNA encoding either the MHC class II/peptide or TCR heterodimers was ligated to DNA coding for murine Ig heavy and light chains. These constructs were subsequently expressed in a baculovirus expression system. Enzyme-linked immunosorbant assays (ELISA) specific for the Ig and polymorphic determinants of either the TCR or MHC fraction of the molecule indicated that infected insect cells secreted approximately 1 .mu.g/ml of soluble, conformationally intact chimeric superdimers. SDS PAGE gel analysis of purified protein showed that expected molecular weight species. The results of flow cytometry demonstrated that the TCR and class II chimeras bound specifically with high avidity to cells bearing their cognate receptors. These superdimers will be useful for studying TCR/MHC interactions, lymphocyte tracking, identifying new antigens, and have possible uses as specific regulators of immune responses.

L19 ANSWER 101 OF 178 USPATFULL
AN 2000:7195 USPATFULL
TI Method for stimulating an immune response utilizing recombinant alphavirus particles
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Chang, Steven M.W., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 6015694 20000118

AI US 1997-931869 19970916 (8)
RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a
continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995,
now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Brusca, John S.
LREP McMasters, David D., Blackburn, Robert P.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10431
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for utilizing
recombinant alphavirus vectors. Also disclosed are compositions and
methods for making and utilizing eukaryotic layered vector initiation
systems.

L19 ANSWER 102 OF 178 USPATFULL
AN 2000:7187 USPATFULL
TI Eukaryotic layered vector initiation systems
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
Driver, David A., San Diego, CA, United States
PA Chiron Viagene, Inc., Emeryville, CA, United States (U.S. corporation)
PI US 6015686 20000118
AI US 1995-404796 19950315 (8)
RLI Continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995,
now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP Seed & Berry, Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10466
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for utilizing

recombinant alphavirus vectors. Also disclosed are compositions and
methods for making and utilizing eukaryotic layered vector initiation
systems.

L19 ANSWER 103 OF 178 USPATFULL
AN 1999:170215 USPATFULL
TI Method and compositions for the **treatment** of autoimmune disease using heat shock proteins
IN Srivastava, Pramod K., Avon, CT, United States
Chandawarkar, Rajiv Y., Akron, OH, United States
PA Fordham University, Bronx, NY, United States (U.S. corporation)
PI US 6007821 19991228
AI US 1997-951789 19971016 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: VanderVegt, F.
Pierre
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 40
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 2004
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to methods and compositions for the **treatment** of autoimmune disease. Specifically, compositions comprising heat shock proteins, including gp96, hsp90, and hsp70, are
disclosed. Immunotherapeutic methods for administering the hsp-containing compositions are disclosed. Furthermore, methods for
preventing rejection of organs transplanted to treat autoimmune disease
are disclosed. The disclosed methods are useful for treating a variety
of autoimmune diseases, including insulin dependent diabetes mellitus.

L19 ANSWER 104 OF 178 USPATFULL
AN 1999:146758 USPATFULL
TI T-cell selective interleukin-4 agonists
IN Shanafelt, Armen B., Moraga, CA, United States
Greve, Jeffrey, Berkeley, CA, United States
Gundel, Robert, Walnut Creek, CA, United States
PA Bayer Corporation, Pittsburgh, PA, United States (U.S. corporation)
PI US 5986059 19991116
AI US 1997-874697 19970613 (8)
PRAI US 1996-19748 19960614 (60)
US 1997-36746 19970127 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Jones, Huw R.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 14 Drawing Page(s)

LN.CNT 2464
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to human IL-4 muteins numbered in accordance with wild-type IL-4 having T cell activating activity, but having reduced endothelial cell activating activity. In particular, the invention is related to human IL-4 muteins wherein the surface-exposed residues of the D helix of the wild-type IL-4 are mutated whereby the resulting mutein causes T cell proliferation, and causes reduced IL-6 secretion from HUVECs, relative to wild-type IL-4. This invention realizes a less toxic IL-4 mutant that allows greater therapeutic use of this interleukin. Further, the invention is directed to IL-4 muteins having single, double and triple mutations represented by the designators R121A, R121D, R121E, R121F, R121H, R1211, R121K, R121N, R121P, R121T, R121W; Y124A, Y124Q, Y124R, Y124S, Y124T; Y124A/S125A, T13D/R121E; and R121T/E122F/Y124Q, when numbered in accordance with wild type IL-4 (His=1). The invention also includes polynucleotides coding for the muteins of the invention, vectors containing the polynucleotides, transformed host cells, pharmaceutical compositions comprising the muteins, and therapeutic methods of **treatment**.

L19 ANSWER 105 OF 178 USPATFULL
AN 1999:146302 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Dulbin, Ireland
Spaulding, Vikki, Billerica, MA, United States
Evans, Cheryl, Germantown, MD, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5985602 19991116
AI US 1996-721925 19960927 (8)
RLI Continuation-in-part of Ser. No. US 1996-701931, filed on 23 Aug 1996, now abandoned which is a continuation-in-part of Ser. No. US 1996-702420, filed on 14 Aug 1996, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique D.
LREP Sprunger, Suzanne A., Brown, Scott A.

CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1599
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 106 OF 178 USPATFULL
AN 1999:145987 USPATFULL
TI Compounds and methods for **treatment** and diagnosis of mycobacterial infections
IN Tan, Paul, Auckland, New Zealand
Skinner, Margot, Auckland, New Zealand
Prestidge, Ross, Auckland, New Zealand
PA Genesis Research and Development Corporation Limited, Parnell, New Zealand (non-U.S. corporation)
PI US 5985287 19991116
AI US 1997-997362 19971223 (8)
RLI Continuation-in-part of Ser. No. US 1997-873970, filed on 12 Jun 1997 which is a continuation-in-part of Ser. No. US 1996-705347, filed on 29 Aug 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Mosher, Mary E.
LREP Sleath, Janet, Speckman, Ann W.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 4862
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides polypeptides comprising an immunogenic portion of a M. vaccae protein and DNA molecules encoding such polypeptides, together with methods for their use in the diagnosis and **treatment** of mycobacterial infection. Methods for enhancing the immune response to an antigen including administration of M. vaccae culture filtrate or delipidated M. vaccae cells are also provided.

L19 ANSWER 107 OF 178 USPATFULL
AN 1999:141912 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Trustees of The University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
PI US 5981505 19991109
WO 9416737 19940804
AI US 1997-979385 19971126 (8)

WO 1994-US899 19940126
19950828 PCT 371 date
19950828 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1993-124962, filed on 21 Sep 1993,
now abandoned And a continuation-in-part of Ser. No. US 1993-93235,
filed on 15 Jul 1993, now abandoned And a continuation of Ser. No. US
1995-495684, filed on 28 Aug 1995, now abandoned which is a
continuation-in-part of Ser. No. US 1993-125012, filed on 21 Sep 1993,
now patented, Pat. No. US 5593972, issued on 14 Jan 1997 which is a
continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993,
now abandoned which is a continuation-in-part of Ser. No. US 1993-8342,
filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 75
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 4084
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of inducing genetic material into cells of an individual and
compositions and kits for practicing the same are disclosed. The methods
comprise the steps of contacting cells of an individual with a
polynucleotide function enhancer and administering to the cells, a
nucleic acid molecule that is free of retroviral particles. The nucleic
acid molecule comprises a nucleotide sequence that encodes a protein
that comprises at least one epitope that is identical or substantially
similar to an epitope of a pathogen antigen or an antigen associated
with a hyperproliferative or autoimmune disease, a protein otherwise
missing from the individual due to a missing, non-functional or
partially functioning gene, or a protein that produces a therapeutic
effect on an individual. Methods of prophylactically and therapeutically
immunizing an individual against HIV are disclosed. Pharmaceutical
compositions and kits for practicing methods of the present invention
are disclosed.

L19 ANSWER 108 OF 178 USPATFULL
AN 1999:141629 USPATFULL
TI Human semaphorin E, secreted proteins and polynucleotides encoding them

IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5981222 19991109
AI US 1997-858834 19970519 (8)
RLI Division of Ser. No. US 1996-702080, filed on 23 Aug 1996, now patented,
Pat. No. US 5654173, issued on 5 Aug 1997
DT Utility
FS Granted
EXNAM Primary Examiner: Feisee, Lila; Assistant Examiner: Kaufman, Claire M.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1801
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 109 OF 178 USPATFULL
AN 1999:136987 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
Agostino, Michael J., Andover, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5976838 19991102
AI US 1997-993228 19971218 (8)
RLI Continuation-in-part of Ser. No. US 1997-781225, filed on 10 Jul 1997,
now abandoned which is a continuation-in-part of Ser. No. US 1996-769100, filed on 18 Dec 1996, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Hamilton, Brook, Smith & Reynolds, P.C.
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 4033
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 110 OF 178 USPATFULL
AN 1999:136986 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
Agostino, Michael J., Andover, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5976837 19991102
AI US 1997-960022 19971029 (8)
RLI Continuation-in-part of Ser. No. US 1997-815047, filed on 14 Mar 1997, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique D.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 3683
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 111 OF 178 USPATFULL
AN 1999:136685 USPATFULL
TI Pretargeting protocols for the enhanced localization of cytotoxins to target sites and cytotoxic combinations useful therefore
IN Fritzberg, Alan R., Edmonds, WA, United States
Abrams, Paul G., Seattle, WA, United States
Reno, John M., Brier, WA, United States
Axworthy, Donald B., Brier, WA, United States
Graves, Scott S., Monroe, WA, United States
Kasina, Sudhakar, Kirkland, WA, United States
PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PI US 5976535 19991102
AI US 1995-468513 19950606 (8)
RLI Continuation of Ser. No. US 1993-163188, filed on 7 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. WO 1993-US5406, filed on 7 Jun 1993 which is a continuation-in-part of Ser. No. US 1992-995381, filed on 23 Dec 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-895588, filed on 9 Jun 1992, now patented, Pat. No. US 5288342
DT Utility

FS Granted
EXNAM Primary Examiner: Cunningham, Thomas M.
LREP Seed and Berry LLP
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 4278
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for targeting cytotoxins to target sites by administration of a combination of conjugates are provided. Novel cytotoxic combinations for use in such methods are also provided.

L19 ANSWER 112 OF 178 USPATFULL
AN 1999:132541 USPATFULL
TI Polynucleotides encoding secreted proteins
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
Racie, Lisa A., Acton, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Merberg, David, Acton, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5972652 19991026
AI US 1997-924838 19970905 (8)
RLI Division of Ser. No. US 1996-628364, filed on 5 Apr 1996, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Mertz, Prema
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1635
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 113 OF 178 USPATFULL
AN 1999:132489 USPATFULL
TI High affinity nucleic acid ligands of cytokines
IN Tasset, Diane, Boulder, CO, United States
Pagratis, Nikos, Boulder, CO, United States
Jayasena, Sumedha, Boulder, CO, United States
Gold, Larry, Boulder, CO, United States
PA NeXstar Pharmaceuticals, Inc., Boulder, CO, United States (U.S. corporation)
PI US 5972599 19991026
AI US 1995-477527 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1991-714131, filed on 10 Jun 1991, now patented, Pat. No. US 5475096 Ser. No. Ser. No. US 1992-931473, filed on 17 Aug 1992, now patented, Pat. No. US 5270163 Ser. No. Ser. No. US 1992-964624, filed on 21 Oct 1992, now patented, Pat. No. US

5496938 And Ser. No. US 1993-117991, filed on 8 Sep 1993, now patented,
Pat. No. US 5660985 , said Ser. No. US 714131 which is a continuation-in-part of Ser. No. US 1990-536428, filed on 11 Jun 1990,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP Swanson & Bratschun LLC
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 5455
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are described for the identification and preparation of high-affinity nucleic acid ligands to cytokines. Included in the
invention are specific nucleic acid ligands to IFN-gamma, IL-4, IL-10,
TNF-alpha, and RANTES.

L19 ANSWER 114 OF 178 USPATFULL
AN 1999:128741 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5969125 19991019
AI US 1996-721924 19960927 (8)
RLI Continuation-in-part of Ser. No. US 1996-686878, filed on 26 Jul 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1574
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 115 OF 178 USPATFULL
AN 1999:128709 USPATFULL
TI Secreted proteins
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
McCoy, John M., Reading, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S.

corporation)
PI US 5969093 19991019
AI US 1997-833823 19970410 (8)
RLI Division of Ser. No. US 1995-514014, filed on 11 Aug 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Prouty, Rebecca E.; Assistant Examiner: Nashed,
Nashaat T.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 16
ECL Exemplary Claim: 6
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1972
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 116 OF 178 USPATFULL
AN 1999:128142 USPATFULL
TI Methods and compounds for the **treatment** of immunologically-mediated psoriasis
IN Watson, James D., Auckland, New Zealand
Tan, Paul L. J., Auckland, New Zealand
PA Genesis Research & Development Corp., Auckland, New Zealand (non-U.S.
corporation)
PI US 5968524 19991019
AI US 1997-997080 19971223 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Devi, S.
LREP Sleath, Janet, Speckman, Ann W.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 6522
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for the **treatment** of skin disorders, including psoriasis, atopic dermatitis, allergic contact dermatitis, alopecia areata and skin cancers are provided, such methods comprising administering multiple doses of a composition having antigenic and/or adjuvant properties. Compositions which may be usefully employed in the inventive methods include inactivated M. vaccae cells, delipidated and deglycolipidated M. vaccae cells, M. vaccae culture filtrate and compounds present in or derived therefrom, together with combinations of such compositions.

L19 ANSWER 117 OF 178 USPATFULL
AN 1999:125029 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States

McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Germantown, MD, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5965693 19991012
AI US 1997-858830 19970519 (8)
RLI Division of Ser. No. US 1996-702080, filed on 23 Aug 1996, now patented,
Pat. No. US 5654173, issued on 5 Aug 1997
DT Utility
FS Granted
EXNAM Primary Examiner: Feisee, Lila; Assistant Examiner: Kaufman, Claire M.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1679
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 118 OF 178 USPATFULL
AN 1999:124733 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Harvard, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
Agostino, Michael J., Andover, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5965397 19991012
AI US 1998-14969 19980128 (9)
RLI Continuation-in-part of Ser. No. US 1997-792511, filed on 31 Jan 1997
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Lahive & Cockfield, LLP, Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 3525
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 119 OF 178 USPATFULL
AN 1999:124724 USPATFULL

TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Germantown, MD, United States
Bowman, Michael, Canton, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5965388 19991012
AI US 1996-721488 19960927 (8)
RLI Continuation-in-part of Ser. No. US 1996-677231, filed on 9 Jul 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1953
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 120 OF 178 USPATFULL
AN 1999:121330 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Carrano, Richard A., Paoli, PA, United States
Wang, Bin, Haidian, China
Weiner, David B., Merion, PA, United States
PA Apollon, Inc., Malvern, PA, United States (U.S. corporation)
The Trustees Of The University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
PI US 5962428 19991005
WO 9526718 19951012
AI US 1996-704701 19960916 (8)
WO 1995-US4071 19950330
19960916 PCT 371 date
19960916 PCT 102(e) date
RLI Continuation of Ser. No. US 221579
DT Utility
FS Granted
EXNAM Primary Examiner: Elliott, George C.; Assistant Examiner: Schwartzman,
Robert
LREP Woodcock Washburn Kurtz Mackiewcz & Norris LLP
CLMN Number of Claims: 42
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 3606
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of introducing genetic material into cells of an individual and

compositions and kits for practicing the same are disclosed. The methods comprise the steps of contacting cells of an individual with a genetic vaccine facilitator and administering to the cells a nucleic acid molecule that is free of retroviral particles. The nucleic acid molecule comprises a nucleotide sequence that encodes a protein that comprises at least one epitope that is identical or substantially similar to an epitope of a pathogen antigen or an antigen associated with a hyperproliferative or autoimmune disease, a protein otherwise missing from the individual due to a missing, non-functional or partially functioning gene, or a protein that produces a therapeutic effect on an individual. Methods of prophylactically and therapeutically immunizing an individual against HIV are disclosed. Pharmaceutical compositions and kits for practicing methods of the present invention are disclosed.

L19 ANSWER 121 OF 178 USPATFULL
AN 1999:121222 USPATFULL
TI Engineered antigen presenting cells and methods for their use
IN Robinson, William S., Burlingame, CA, United States
PA Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)
PI US 5962320 19991005
AI US 1997-888360 19970703 (8)
RLI Continuation-in-part of Ser. No. US 663157
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1364
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Autologous, heterologous or xenogeneic primary cells or cell lines are genetically modified ex vivo to render the cells capable of processing and presenting selected antigens to cells of the immune system of a subject, and to express different HLA molecules for matching to the HLA specificity of the subject. The cells are also modified to express immunoregulatory molecules for directing the immune response of the subject. The cells and cell lines are used in methods to treat infectious diseases or cancer, or to prevent infectious disease by

inoculation into a host to activate T cells and induce an antigen-specific immune response, and in assays of the cytolytic activity of a subject's T cells. The cells can also be used to suppress an unwanted immune response of a subject to a selected antigen where the cells lack expression of a costimulation molecule needed for T cell activation.

L19 ANSWER 122 OF 178 USPATFULL
AN 1999:117298 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
Racie, Lisa A., Acton, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Woburn, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5958726 19990928
AI US 1997-867680 19970602 (8)
RLI Continuation-in-part of Ser. No. US 1996-635311, filed on 19 Apr 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique D.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1766
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 123 OF 178 USPATFULL
AN 1999:117243 USPATFULL
TI Methods and compositions for regulating T cell subsets by modulating transcription factor activity
IN Glimcher, Laurie H., West Newton, MA, United States
Ho, I-Cheng, Newton, MA, United States
PA Presidents and Fellows of Harvard College, Cambridge, MA, United States (U.S. corporation)
PI US 5958671 19990928
AI US 1996-636602 19960423 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Campell, Bruce R.; Assistant Examiner: Priebe, Scott D.
LREP Lahive & Cockfield, LLP, DeConti, Jr., Giulio A., Kara, Catherine J.

CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2803
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for modulating production of a T helper type 2 (Th2)-associated cytokine, in particular interleukin-4, by modulating the activity of a transcription factor, in particular the proto-oncoprotein c-Maf, that regulates expression of the Th2-associated cytokine gene are disclosed. Methods for modulating development of T helper type 1 (Th1) or T helper type 2 (Th2) subsets in a subject using agents that modulate transcription factor activity are also disclosed. The methods of the invention can further involve use of agents that modulate the activity of additional transcription factors that contribute to the regulation of Th1- or Th2-associated cytokines, such as a Nuclear Factor of Activated T cells (NF-AT) protein and/or an AP-1 family protein. Compositions for modulating Th2-associated cytokine production, recombinant expression vectors and host cells, as well as screening assays to identify agents that modulate c-Maf activity, are also disclosed.

L19 ANSWER 124 OF 178 USPATFULL
AN 1999:113762 USPATFULL
TI Substituted 2-(2,6-dioxo-3-fluoropiperidin-3-yl)-isoindolines and method of reducing inflammatory cytokine levels
IN Muller, George W., Bridgewater, NJ, United States
Stirling, David I., Branchburg, NJ, United States
Chen, Roger Shen-Chu, Edison, NJ, United States
Man, Hon-Wah, Neshanic Station, NJ, United States
PA Celgene Corporation, Warren, NJ, United States (U.S. corporation)
PI US 5955476 19990921
AI US 1998-42274 19980313 (9)
RLI Continuation-in-part of Ser. No. US 1997-976140, filed on 18 Nov 1997, now patented, Pat. No. US 5874448
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Rao, Deepak R.
LREP Mathews, Collins, Shepherd & Gould, P.A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1022
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB 1-Oxo-2-(2,6-dioxo-3-fluoropiperidin-3-yl)isoindolines and 1,3-dioxo-2-(2,6-dioxo-3-fluoropiperidin-3-yl)isoindolines reduce the levels of inflammatory cytokines such as **TNF**.alpha. in a mammal. A typical embodiment is 1,3-dioxo-2-(2,6-dioxo-3-fluoropiperidin-3 -yl)-isoindoline.

L19 ANSWER 125 OF 178 USPATFULL
AN 1999:102691 USPATFULL
TI Polynucleotides encoding secreted proteins
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5945302 19990831
AI US 1997-783395 19970113 (8)
RLI Continuation-in-part of Ser. No. US 1996-628364, filed on 5 Apr 1996, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Mertz, Prema
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1550
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 126 OF 178 USPATFULL
AN 1999:96351 USPATFULL
TI DNA vaccination for induction of suppressive T cell response
IN Steinman, Lawrence, Palo Alto, CA, United States
Waisman, Ari, Tel-Aviv, Israel
PA The Board of Trustees of The Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)
PI US 5939400 19990817
AI US 1996-606639 19960226 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Crouch, Deborah; Assistant Examiner: Martin, Jill D.
LREP Bozicevic, Field & Francis LLP, Sherwood, Pamela J.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 952
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A pro-inflammatory T response is specifically prevented by the injection into a recipient of DNA encoding the variable region of a T cell

receptor. In response to the vaccination, T cells expressing the variable region produce Th2 cytokines, including IL-4. A pro-inflammatory T cell response directed to an autoantigen is shown to be suppressed by DNA vaccination. The suppressive vaccination further reduced the inflammatory effect of T cells reactive against epitopes of the autoantigen not recognized by the variable region used for vaccination.

L19 ANSWER 127 OF 178 USPATFULL
AN 1999:96020 USPATFULL
TI Materials and methods for detection and **treatment** of immune system dysfunctions
IN Clare-Salzler, Michael, Gainesville, FL, United States
PA University of Florida, Gainesville, FL, United States (U.S. corporation)
PI US 5939069 19990817
AI US 1996-701928 19960823 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David
LREP Saliwanchik, Lloyd & Saliwanchik
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 717
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The subject invention concerns novel materials and methods for the **treatment** and/or prevention of autoimmune disease. In a specific embodiment, elevated production of prostaglandin synthase-2 (PGS-2) is correlated with autoimmune dysfunction.

L19 ANSWER 128 OF 178 USPATFULL
AN 1999:92287 USPATFULL
TI Gene **therapy** for effector cell regulation
IN Dow, Steve W., Denver, CO, United States
Elmslie, Robyn E., Denver, CO, United States
Potter, Terence A., Denver, CO, United States
PA National Jewish Medical & Research Center, Denver, CO, United States
(U.S. corporation)
PI US 5935568 19990810
AI US 1995-580806 19951229 (8)
RLI Continuation-in-part of Ser. No. US 1995-446918, filed on 18 May 1995, now patented, Pat. No. US 5705151 And a continuation-in-part of Ser. No. US 1995-484169, filed on 7 Jun 1995, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Stanton, Brian R.; Assistant Examiner: Hauda, Karen M.
LREP Ross P.C., Sheridan
CLMN Number of Claims: 28

ECL Exemplary Claim: 1,3,5
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2705
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a nucleic acid-based therapeutic composition to treat an animal with disease by controlling the activity of effector cells, including T cells, macrophages, monocytes and/or natural killer cells, in the animal. Therapeutic compositions of the present invention include superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules, depending upon the disease being treated. The present invention also relates to an adjuvant for use with nucleic acid-based vaccines. Adjuvant compositions of the present invention include an immunogen combined with superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules.

L19 ANSWER 129 OF 178 USPATFULL
AN 1999:81543 USPATFULL
TI Soluble lymphotoxin-.beta. receptors and anti-lymphotoxin receptor and ligand **antibodies** as therapeutic agents for the **treatment** of immunological disease
IN Browning, Jeffrey L., Brookline, MA, United States
Benjamin, Christopher D., Beverly, MA, United States
Hochman, Paula S., Brookline, MA, United States
PA Biogen, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5925351 19990720
AI US 1995-505606 19950721 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Bansal, Geetha P.
LREP Biogen, Inc., Flynn, Kerry A.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1,15
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1968
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to compositions and methods comprising "lymphotoxin-.beta. receptor blocking agents", which block lymphotoxin-.beta. receptor signalling. Lymphotoxin-.beta. receptor blocking agents are useful for treating lymphocyte-mediated immunological diseases, and more particularly, for inhibiting Th1 cell-mediated immune responses. This invention relates to soluble forms

of the lymphotoxin-.beta. receptor extracellular domain that act as
lymphotoxin-.beta. receptor blocking agents. This invention also relates
to the use of **antibodies** directed against either the
lymphotoxin-.beta. receptor or its ligand, surface lymphotoxin, that act
as lymphotoxin-.beta. receptor blocking agents. A novel screening method
for selecting soluble receptors, **antibodies** and other agents
that block LT-.beta. receptor signalling is provided.

L19 ANSWER 130 OF 178 USPATFULL
AN 1999:78850 USPATFULL
TI Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor
antibodies
IN Deo, Yashwant M., Audubon, PA, United States
Graziano, Robert, Frenchtown, NJ, United States
Keler, Tibor, Ottsville, PA, United States
PA Medarex, Inc., Annandale, NJ, United States (U.S. corporation)
PI US 5922845 19990713
AI US 1996-678194 19960711 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Toni R.; Assistant Examiner: Bansal, Geetha
P.
LREP Lahive & Cockfield, LLP, DeConiti, Jr., Giulio A., Remillard, Jane E.
CLMN Number of Claims: 24
ECL Exemplary Claim: 1,13
DRWN 15 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 2127
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor
antibodies and methods of use are provided.

L19 ANSWER 131 OF 178 USPATFULL
AN 1999:75759 USPATFULL
TI Low affinity human **IL-12** beta2 receptor
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5919903 19990706
AI US 1997-914520 19970819 (8)
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701 19950801 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1531

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** receptor complex produced on the surface of a non-human mammalian cell and free from other human proteins, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity. A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein or the recombinant human **IL-12** receptor complex, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein or the complex expressed on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L19 ANSWER 132 OF 178 USPATFULL
AN 1999:56462 USPATFULL
TI Pharmaceutical angiostatic dipeptide compositions and method of use thereof
IN Green, Lawrence R., Tacoma, WA, United States
Blasecki, John W., Woodinville, WA, United States
PA Cytran, Inc., Kirkland, WA, United States (U.S. corporation)
PI US 5902790 19990511
AI US 1996-614764 19960313 (8)
RLI Continuation-in-part of Ser. No. US 1995-538701, filed on 3 Oct 1995, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner: Celsa, Bennett
LREP Townsend & Townsend & Crew LLP
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1040
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods of inhibiting neovascularization in a subject by administering to the subject a pharmaceutical preparation of R'-Glu-Trp-R".

L19 ANSWER 133 OF 178 USPATFULL
AN 1999:43610 USPATFULL
TI **Treatment** of arthritic disease induced by infectious agents
IN DeLuca, Hector F., Deerfield, WI, United States
Cantorna, Margherita T., Middleton, WI, United States
Hayes, Colleen E., Madison, WI, United States

PA Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)
PI US 5891865 19990406
AI US 1996-726894 19961004 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Criares, Theodore J.
LREP Quarles & Brady
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 708
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of treating **arthritis** symptoms induced by an infectious agent of an **arthritis** patient comprising administering to an **arthritis** patient an amount of vitamin D compound effective to reduce symptoms and observing a reduction in symptoms is disclosed.

L19 ANSWER 134 OF 178 USPATFULL
AN 1999:43184 USPATFULL
TI Membrane-bound cytokine compositions comprising GM=CSF and methods of modulating an immune response using same
IN Hoo, William Soo, Carlsbad, CA, United States
PA The Immune Response Corporation, Carlsbad, CA, United States (U.S. corporation)
PI US 5891432 19990406
AI US 1997-902516 19970729 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Spector, Lorraine
LREP Campbell & Flores LLP
CLMN Number of Claims: 24
ECL Exemplary Claim: 1,13
DRWN 9 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1917
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule such as GM-CSF operatively fused to a heterologous membrane attachment domain. Non-**antibody** immunomodulatory molecules useful in the invention include immunostimulatory and immunosuppressive molecules such as cytokines. In one embodiment, the invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain and, additionally, a disease-associated antigen or immunogenic epitope thereof. Further provided by the invention are methods of modulating an immune response against a disease-associated

antigen by administering to an individual a cellular vaccine having a
membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane
attachment domain.

L19 ANSWER 135 OF 178 USPATFULL
AN 1998:162547 USPATFULL
TI Protein kinase inhibitor
IN Sriram, Subramaniam, Nashville, TN, United States
Bright, John, Nashville, TN, United States
Nag, Bishwajit, Fremont, CA, United States
Sharma, Somesh D., Los Altos, CA, United States
PA Natpro, Inc., Union City, CA, United States (U.S. corporation)
PI US 5854285 19981229
AI US 1997-825662 19970403 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: MacMillan, Keith D.
LREP Fish & Richardson P.C.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 265
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A compound of the formula I ##STR1## wherein A and C are independently
H, alkyl of 1-6 carbon atoms, hydroxy, or alkoxy of 1-6 carbon atoms;

B is hydroxy or alkoxy of 1-6 carbon atoms; and

Y is cyano, ##STR2##

--C(NR.sub.1 R.sub.2).dbd.C(CN).sub.2 ;

wherein X=O or S, and R.sub.1 and R.sub.2 are independently H, benzyl,
--CH(CH.sub.3), C.sub.6 H.sub.5

--(CH.sub.2).sub.n C.sub.6 H.sub.6, phenyl; --CO.sub.2 R;

n=2-4; R is lower alkyl of 1-6 carbon atoms

is used for treating inflammation and immunological diseases.

L19 ANSWER 136 OF 178 USPATFULL
AN 1998:160106 USPATFULL
TI **Antibodies** to receptors for human interleukin-12
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5852176 19981222
AI US 1997-915495 19970820 (8)
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701 19950801 (60)
DT Utility

FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1381
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB **Antibodies** to human **IL-12** beta 2 receptor protein or an **IL-12** receptor complex, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity.

L19 ANSWER 137 OF 178 USPATFULL
AN 1998:150739 USPATFULL
TI Alphavirus vector constructs
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Ibanez, Carlos E., San Diego, CA, United States
Chang, Stephen M. W., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
Driver, David A., San Diego, CA, United States
Belli, Barbara A., San Diego, CA, United States
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 5843723 19981201
AI US 1996-739167 19961030 (8)
RLI Continuation of Ser. No. US 1995-404796, filed on 20 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP McMasters, David D., Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 47
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10318
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and method,, for utilizing recombinant alphavirus vectors.

L19 ANSWER 138 OF 178 USPATFULL
AN 1998:147252 USPATFULL
TI DNA encoding receptors for the beta-2 chain of human **IL-12**
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States

Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5840530 19981124
AI US 1996-685118 19960723 (8)
PRAI US 1995-1701 19950801 (60)
US 1996-18674 19960530 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1424
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L19 ANSWER 139 OF 178 USPATFULL
AN 1998:143936 USPATFULL
TI Complexes comprising a nucleic acid bound to a cationic polyamine having an endosome disruption agent
IN Boutin, Raymond H., Thornton, PA, United States
PA American Home Products Corporation, Madison, NJ, United States (U.S. corporation)
PI US 5837533 19981117
AI US 1994-314060 19940928 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Crouch, Deborah
LREP Howson and Howson
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3984
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A multifunctional molecular complex for the transfer of a nucleic acid composition to a target cell is provided which comprises in any functional combination: A) said nucleic acid composition; and B) a transfer moiety comprising 1) one or more cationic polyamine components bound to said nucleic acid composition, each comprising from three to

twelve nitrogen atoms; 2) one or more endosome membrane disruption promoting components attached to at least one nitrogen atom of at least one of said polyamine components, through an alkyl, carboxamide, carbamate, thiocarbamate, or carbamoyl bridging group, comprising a) at least one lipophilic long chain alkyl group, b) a fusogenic peptide comprising spike glycoproteins of enveloped animal viruses, or c) cholic acid or cholesteryl or derivatives; and optionally 3) one or more receptor specific binding components which are ligands for natural receptors of said target cell, attached through an alkyl, carboxamide, carbamate, thiocarbamate, or carbamoyl bridging group to either i) a further nitrogen atom of at least one of said polyamine components to which said one or more endosome membrane disruption promoting components is attached, or ii) a nitrogen atom of at least one further polyamine component which does not have attached thereto any endosome membrane disruption promoting component. Also provided are the transfer moiety alone, or in combination with the nucleic acid composition as a self-assembling combination, and the use of these compositions in methods for transfering nucleic acid compositions to cells or to cells of individuals, for immunizing individuals against a pathogen or disease, and for treating an individual with a disease.

L19 ANSWER 140 OF 178 USPATFULL
AN 1998:143894 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5837490 19981117
AI US 1996-739775 19961030 (8)
RLI Continuation-in-part of Ser. No. US 1996-721923, filed on 27 Sep 1996, now abandoned which is a continuation-in-part of Ser. No. US 1996-664596, filed on 17 Jun 1996
DT Utility
FS Granted

EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Sprunger, Ph.D., Suzanne A., Brown, Scott A.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1647
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 141 OF 178 USPATFULL
AN 1998:138853 USPATFULL
TI Chemokine binding protein and methods of use therefor
IN McFadden, Grant, Edmonton, Canada
Lucas, Alexandra, Edmonton, Canada
PA The John P. Robarts Institute, London, Canada (non-U.S. corporation)
PI US 5834419 19981110
AI US 1996-634924 19960419 (8)
RLI Continuation-in-part of Ser. No. US 1995-424850, filed on 19 Apr 1995,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Basham, Daryl A.
LREP Fish & Richardson, P.C.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1037
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method of use for a novel type I
chemokine binding protein encoded by poxviruses and having amino acid
sequence homology with the myxoma virus T7 interferon-.gamma. receptor
homolog against disease syndromes associated with acute or chronic
dysregulated inflammatory responses.

L19 ANSWER 142 OF 178 USPATFULL
AN 1998:135197 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Bowman, Michael, Canton, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5831056 19981103
AI US 1996-721746 19960927 (8)

RLI Continuation-in-part of Ser. No. US 1996-659224, filed on 7 Jun 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Sprunger, Suzanne A., Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1546
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 143 OF 178 USPATFULL
AN 1998:135151 USPATFULL
TI Human receptor for interleukin-12
IN Chua, Anne On, Wayne, NJ, United States
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5831007 19981103
AI US 1995-419652 19950411 (8)
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented,
Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US
1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 1937
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor
cDNAs and protein and uses therefore. The Interleukin-12 receptor is
shown to be a member of the cytokine receptor superfamily and has a high
homology to human gp130.

L19 ANSWER 144 OF 178 USPATFULL
AN 1998:131564 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5827688 19981027

AI US 1996-738367 19961025 (8)
RLI Continuation-in-part of Ser. No. US 1996-721926, filed on 27 Sep 1996,
now abandoned which is a continuation-in-part of Ser. No. US 1996-664596, filed on 17 Jun 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Hendricks, Keith D.; Assistant Examiner: Longton,
Enrique D.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1703
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 145 OF 178 USPATFULL
AN 1998:128265 USPATFULL
TI Substituted amino alcohol compounds
IN Klein, J. Peter, Vashon, WA, United States
Underiner, Gail E., Brier, WA, United States
Kumar, Anil M., Seattle, WA, United States
PA Cell Therapeutics, Inc., Seattle, WA, United States (U.S. corporation)
PI US 5824677 19981020
AI US 1995-474816 19950607 (8)
RLI Division of Ser. No. US 1994-303842, filed on 8 Sep 1994, now patented,
Pat. No. US 5641783 which is a continuation-in-part of Ser. No. US
1993-152650, filed on 12 Nov 1993, now patented, Pat. No. US 5801181 And
Ser. No. US 1993-164081, filed on 8 Dec 1993, now patented, Pat. No. US
5470878 , said Ser. No. US -152650 And Ser. No. US -164081 , each
Ser. No. US - which is a continuation-in-part of Ser. No. US
1993-40820, filed on 31 Mar 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Cebulak, Mary
C.
LREP McDermott, Will & Emery, Faciszewski, Esq., Stephen
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 120 Drawing Figure(s); 89 Drawing Page(s)
LN.CNT 3136
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are compounds having a straight or branched aliphatic
hydrocarbon structure of formula I: ##STR1## In formula I, n is an
integer from one to four and m is an integer from four to twenty.
Independently, R.sub.1 and R.sub.2 are hydrogen, a straight or branched

chain alkyl, alkenyl or alkynyl of up to twenty carbon atoms in length
or --(CH.sub.2).sub.w R.sub.5. If R.sub.1 or R.sub.2 is --(CH.sub.2).sub.w R.sub.5, w may be an integer from one to twenty and
R.sub.5 may be an hydroxyl, halo, C.sub.1-8 alkoxyl group or a substituted or unsubstituted carbocycle or heterocycle. Alternatively,
R.sub.1 and R.sub.2 may jointly form a substituted or unsubstituted,
saturated or unsaturated heterocycle having from four to eight carbon
atoms, N being a hetero atom of the resulting heterocyle. R.sub.3 may be
either hydrogen or C.sub.13. In the compounds, a total sum of carbon
atoms comprising R.sub.1 or R.sub.2, (CH.sub.2).sub.n and (CH.sub.2).sub.m does not exceed forty. R.sub.4 is a hetorocycle
comprising a substituted or unsubstituted, oxidized or reduced ring
system, the ring system having a single ring or two to three fused
rings, a ring comprising from three to seven ring atoms. The disclosed
compounds are effective agents to inhibit undesirable responses to cell
stimuli.

L19 ANSWER 146 OF 178 USPATFULL
AN 1998:128130 USPATFULL
TI Shigella vector for delivering DNA to a mammalian cell
IN Branstrom, Arthur A., Rockville, MD, United States
Sizemore, Donata R., Gaithersburg, MD, United States
Sadoff, Jerald C., Washington, DC, United States
PA The United States of America as represented by the Secretary of the
Army, Washington, DC, United States (U.S. government)
PI US 5824538 19981020
AI US 1995-523855 19950906 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Lankford, Jr., Leon B.; Assistant Examiner: Tate,
Christopher R.
LREP Harris, Charles H., Moran, John Francis
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1304
AB We describe a bacterial delivery system for the delivery of DNA and
antigens into cells. We constructed an attenuated bacterial vector which
enters mammalian cells and ruptures delivering functional plasmid DNA,
such as a mammalian expression plasmid, and antigens into the cell
cytoplasm. This Shigella vector was designed to deliver DNA to colonic

surfaces, thus opening the possibility of oral and other mucosal DNA
immunization and gene **therapy** strategies. The attenuated
Shigella is also useful as a vaccine for reducing disease symptoms
caused by Shigella.

L19 ANSWER 147 OF 178 USPATFULL
AN 1998:122388 USPATFULL
TI Genetic immunization
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United
States (U.S. corporation)
The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
PI US 5817637 19981006
AI US 1997-783818 19970113 (8)
RLI Continuation of Ser. No. US 1993-125012, filed on 21 Sep 1993, now
patented, Pat. No. US 5593972, issued on 14 Jan 1997 which is a
continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993,
now abandoned which is a continuation-in-part of Ser. No. US 1993-8342,
filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 3641
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of prophylactic and therapeutic immunization of an individual
against pathogen infection, diseases associated with hyperproliferative
cells and autoimmune diseases are disclosed. The methods comprise the
steps of administering to cells of an individual, a nucleic acid
molecule that comprises a nucleotide sequence that encodes a protein
which comprises at least one epitope that is identical or substantially
similar to an epitope of a pathogen antigen, a hyperproliferative cell
associated protein or a protein associated with autoimmune disease
respectively. In each case, nucleotide sequence is operably linked to
regulatory sequences to enable expression in the cells. The nucleic acid
molecule is free of viral particles and capable of being expressed in

said cells. The cells may be contacted cells with a cell stimulating
agent. Methods of prophylactically and therapeutically immunizing an
individual against HIV are disclosed. Pharmaceutical compositions and
kits for practicing methods of the present invention are disclosed.

L19 ANSWER 148 OF 178 USPATFULL
AN 1998:119004 USPATFULL
TI Eukaryotic layered vector initiation systems
IN Dubensky, Jr., Thomas W., P.O. Box 675205, Rancho Sante Fe, CA, United
States 92067
Polo, John M., 1222 Reed Ave., Number 4, San Diego, CA, United States
92109
Jolly, Douglas J., 277 Hillcrest Dr., Leucadia, CA, United States 92024
Driver, David A., 5142 Biltmore St., San Diego, CA, United States 92117
PI US 5814482 19980929
AI US 1996-739158 19961030 (8)
RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a
continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995,
now abandoned which is a continuation-in-part of Ser. No. US
1994-348472, filed on 30 Nov 1994, now abandoned which is a
continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
now abandoned which is a continuation-in-part of Ser. No. US
1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP Seed & Berry, Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10431
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for utilizing
recombinant alphavirus vectors. Also disclosed are compositions and
methods for making and utilizing eukaryotic layered vector initiation
systems.

L19 ANSWER 149 OF 178 USPATFULL
AN 1998:111796 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States

Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5807709 19980915
AI US 1996-721798 19960927 (8)
RLI Continuation-in-part of Ser. No. US 1996-664596, filed on 17 Jun 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Sprunger, Suzanne A., Brown, Scott A.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1492
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 150 OF 178 USPATFULL
AN 1998:111791 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Brookline, MA, United States
Spaulding, Vikki, Billerica, MA, United States
Bowman, Michael, Canton, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5807703 19980915
AI US 1996-664596 19960617 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Longton, Enrique
D.
LREP Sprunger, Ph.D., Suzanne A., Brown, Scott A.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2492
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 151 OF 178 USPATFULL
AN 1998:95405 USPATFULL
TI Secreted protein, BA3.1, and polynucleotides encoding same
IN Bowman, Michael, 50 Aldrich Rd., Canton, MA, United States 02021
PI US 5792628 19980811

AI US 1997-818163 19970314 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Grimes, Eric; Assistant Examiner: Longton, Enrique D.
LREP Brown, Scott A.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1443
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel secreted protein, BA3.1, is disclosed. Polynucleotides encoding
BA3.1 are also provided.

L19 ANSWER 152 OF 178 USPATFULL
AN 1998:91872 USPATFULL
TI Alphavirus structural protein expression cassettes
IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
Polo, John M., San Diego, CA, United States
Ibanez, Carlos E., San Diego, CA, United States
Chang, Stephen M. W., San Diego, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
Driver, David A., San Diego, CA, United States
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 5789245 19980804
AI US 1996-741881 19961030 (8)
RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a
continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995,
now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
LREP McMasters, David D., Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10270
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for utilizing
recombinant alphavirus vectors. Also disclosed are compositions and
methods for making and utilizing eukaryotic layered vector initiation
systems.

L19 ANSWER 153 OF 178 USPATFULL
AN 1998:88944 USPATFULL
TI Secreted proteins and polynucleotides encoding them

IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5786465 19980728
AI US 1996-721489 19960927 (8)
RLI Continuation-in-part of Ser. No. US 1996-686878, filed on 26 Jul 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1570
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 154 OF 178 USPATFULL
AN 1998:79344 USPATFULL
TI Method for preparing substituted amino alcohol compounds
IN Klein, J. Peter, Vashon, WA, United States
Underiner, Gail E., Brier, WA, United States
Kumar, Anil M., Seattle, WA, United States
PA Cell Therapeutics, Inc., Seattle, WA, United States (U.S. corporation)
PI US 5777117 19980707
AI US 1995-472569 19950607 (8)
RLI Division of Ser. No. US 1994-303842, filed on 8 Sep 1994 which is a
continuation-in-part of Ser. No. US 1993-152650, filed on 12 Nov 1993
And Ser. No. US 1993-164081, filed on 8 Dec 1993 which is a continuation-in-part of Ser. No. US 1993-40820, filed on 31 Mar 1993,
now abandoned , said Ser. No. US -152650 which is a continuation-in-part of Ser. No. US -40820
DT Utility
FS Granted
EXNAM Primary Examiner: Dees, Jose G.; Assistant Examiner: Cebulak, Mary C.
LREP McDermott, Will & Emery
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 118 Drawing Figure(s); 92 Drawing Page(s)
LN.CNT 3153
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a process for preparing compounds having a straight or
branched aliphatic hydrocarbon structure of formula I:
##STR1## In

formula I, n is an integer from one to four and m is an integer from four to twenty. Independently, R.sub.1 and R.sub.2 are hydrogen, a straight or branched chain alkyl, alkenyl or alkynyl of up to twenty carbon atoms in length or --(CH.sub.2).sub.w R.sub.5. If R.sub.1 or R.sub.2 is --(CH.sub.2).sub.w R.sub.5, w may be an integer from one to twenty and R.sub.5 may be an hydroxyl, halo, C.sub.1-8 alkoxyl group or a substituted or unsubstituted carbocycle or heterocycle. Alternatively, R.sub.1 and R.sub.2 may jointly form a substituted or unsubstituted, saturated or unsaturated heterocycle having from four to eight carbon atoms, N being a hetero atom of the resulting heterocyle. R.sub.3 may be either hydrogen or C.sub.1-3. In the compounds, a total sum of carbon atoms comprising R.sub.1 or R.sub.2, (CH.sub.2).sub.n and (CH.sub.2).sub.m does not exceed forty. R.sub.4 is a terminal moiety comprising a substituted or unsubstituted, oxidized or reduced ring system, the ring system having a single ring or two to three fused rings, a ring comprising from three to seven ring atoms. The disclosed compounds are effective agents to inhibit undesirable responses to cell stimuli.

L19 ANSWER 155 OF 178 USPATFULL
AN 1998:51651 USPATFULL
TI Substituted amino alcohol compounds
IN Klein, J. Peter, Vashon, WA, United States
Underiner, Gail E., Brier, WA, United States
Kumar, Anil M., Seattle, WA, United States
PA Cell Therapeutics, Inc., Seattle, WA, United States (U.S. corporation)
PI US 5750575 19980512
AI US 1995-475721 19950607 (8)
RLI Division of Ser. No. US 1994-303842, filed on 8 Sep 1994, now patented, Pat. No. US 5641783 which is a continuation-in-part of Ser. No. US 1993-152650, filed on 12 Nov 1993 And a continuation-in-part of Ser. No. US 1993-164081, filed on 8 Dec 1993, now patented, Pat. No. US 5470878 which is a continuation-in-part of Ser. No. US 1993-40820, filed on 31 Mar 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Dees, Jose G.; Assistant Examiner: Cebulak, M.

LREP McDermott, Will & Emery, Faciszewski, Esq., Stephen
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 115 Drawing Figure(s); 90 Drawing Page(s)
LN.CNT 3115
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are compounds having a straight or branched aliphatic hydrocarbon structure of formula I: ##STR1## In formula I, n is an integer from one to four and m is an integer from four to twenty. Independently, R.sub.1 and R.sub.2 are hydrogen, a straight or branched chain alkyl, alkenyl or alkynyl of up to twenty carbon atoms in length or --(CH.sub.2).sub.w R.sub.5. If R.sub.1 or R.sub.2 is --(CH.sub.2).sub.w R.sub.5, w may be an integer from one to twenty and R.sub.5 may be an hydroxyl, halo, C.sub.1-8 alkoxyl group or a substituted or unsubstituted carbocycle or heterocycle. Alternatively, R.sub.1 and R.sub.2 may jointly form a substituted or unsubstituted, saturated or unsaturated heterocycle having from four to eight carbon atoms, N being a hetero atom of the resulting heterocyle. R.sub.3 may be either hydrogen or C.sub.1-3. In the compounds, a total sum of carbon atoms comprising R.sub.1 or R.sub.2, (CH.sub.2).sub.n and (CH.sub.2).sub.m does not exceed forty. R.sub.4 is a carbocycle comprising a substituted or unsubstituted ring system, the ring system having a single ring or two fused rings, a ring comprising from three to seven ring atoms. The disclosed compounds are effective agents to inhibit undesirable responses to cell stimuli.

L19 ANSWER 156 OF 178 USPATFULL
AN 1998:39510 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Carrano, Richard A., Paoli, PA, United States
Wang, Bin, Beijing, China
Weiner, David B., Merion, PA, United States
PA Apollon, Inc., Malvern, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
PI US 5739118 19980414
AI US 1994-221579 19940401 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Rories, Charles C. P.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris, LLP
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 3405

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of introducing genetic material into cells of an individual and compositions and kits for practicing the same are disclosed. The methods comprise the steps of contacting cells of an individual with a genetic vaccine facilitator and administering to the cells, a nucleic acid molecule that is free of retroviral particles. The nucleic acid molecule comprises a nucleotide sequence that encodes a protein that comprises at least one epitope that is identical or substantially similar to an epitope of a pathogen antigen or an antigen associated with a hyperproliferative or autoimmune disease, a protein otherwise missing from the individual due to a missing, non-functional or partially functioning gene, or a protein that produce a therapeutic effect on an individual. Methods of prophylactically and therapeutically immunizing an individual against HIV are disclosed. Pharmaceutical compositions and kits for practicing methods of the present invention are disclosed.

L19 ANSWER 157 OF 178 USPATFULL
AN 1998:28196 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Brookline, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5728819 19980317
AI US 1996-691641 19960802 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1864
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 158 OF 178 USPATFULL
AN 1998:25211 USPATFULL

TI Cytokine regulatory agents and methods of use in pathologies and
conditions associated with altered cytokine levels
IN Girten, Beverly E., San Diego, CA, United States
Andalibi, Ali, San Diego, CA, United States
Basu, Amaresh, San Diego, CA, United States
Fagan, Patrick, Escondido, CA, United States
Houghten, Richard A., Del Mar, CA, United States
Loullis, Costas C., Cardiff, CA, United States
Omholt, Paul, San Diego, CA, United States
Tuttle, Ronald R., Escondido, CA, United States
Suto, Mark J., San Diego, CA, United States
Weber, Patricia A., Stevensville, MT, United States
PA Trega Biosciences, Inc., San Diego, CA, United States (U.S. corporation)
PI US 5726156 19980310
AI US 1995-527056 19950912 (8)
RLI Continuation-in-part of Ser. No. US 1995-484262, filed on 7 Jun 1995,
now abandoned which is a continuation-in-part of Ser. No. US
1995-400983, filed on 6 Mar 1995
DT Utility
FS Granted
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner:
Delacroix-Muirheid, C.
LREP Campbell & Flores LLP
CLMN Number of Claims: 31
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1873
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel peptides that are potent cytokine
regulatory agents. In addition, the present invention relates to
pharmaceutical compositions comprising a pharmaceutically acceptable
carrier and a cytokine regulatory agent. Administration of such a
cytokine regulatory agent to a subject can enhance or restrain cytokine
activity. Thus, the present invention provides a method of regulating
cytokine activity in a subject having a condition characterized by
aberrant or altered cytokine activity. The invention also provides
methods of treating such conditions, including, for example, disuse
deconditioning, diseases mediated by nitric oxide and cytokines, adverse
drug reactions, obesity, septic shock, and adverse side effects due to
cancer chemotherapy or occurring as in response to organ
transplantation.

L19 ANSWER 159 OF 178 USPATFULL
AN 1998:22079 USPATFULL
TI Secreted proteins and polynucleotides encoding them

IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5723315 19980303
AI US 1996-702344 19960823 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2437
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 160 OF 178 USPATFULL
AN 1998:4755 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewskbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Evans, Cheryl, Brookline, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5708157 19980113
AI US 1996-686878 19960726 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Kaufman, Claire M.
LREP Brown, Scott A., Sprunger, Suzanne A., DesRosier, Thomas J.
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3204
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 161 OF 178 USPATFULL
AN 1998:4432 USPATFULL
TI DNA sequences and secreted proteins encoded thereby
IN Jacobs, Kenneth, Newton, MA, United States
Kelleher, Kerry, Marlborough, MA, United States
Carlin, McKeough, Cambridge, MA, United States
McCoy, John M., Reading, MA, United States

PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5707829 19980113
AI US 1995-514014 19950811 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Nashed, Nashaat T.
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 44
ECL Exemplary Claim: 44
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1689
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 162 OF 178 USPATFULL
AN 1998:1445 USPATFULL
TI Gene **therapy** for T cell regulation
IN Dow, Steve W., Denver, CO, United States
Elmslie, Robyn E., Denver, CO, United States
PA National Jewish Center for Immunology & Respiratory Medicine, Denver, CO, United States (U.S. corporation)
PI US 5705151 19980106
AI US 1995-446918 19950518 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Chambers, Jasemine C.; Assistant Examiner: Hauda, Karen M.
LREP Sheridan Ross P.C.
CLMN Number of Claims: 52
ECL Exemplary Claim: 1,28
DRWN 10 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2206
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a nucleic acid-based therapeutic composition to treat an animal with disease by controlling the activity of effector cells, including T cells, macrophages, monocytes and/or natural killer cells, in the animal. The present invention also relates to methods of gene **therapy** involving different modes of administration of a therapeutic composition to treat animals with different types of diseases. Also included in the present invention are recombinant molecules for use in a therapeutic composition and recombinant cells useful as a tumor vaccine. Therapeutic compositions of the present invention include superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule, depending upon the disease being treated.

L19 ANSWER 163 OF 178 USPATFULL
AN 97:117939 USPATFULL
TI Methods and compositions for inhibiting production of replication competent virus
IN Klump, Wolfgang M., Del Mar, CA, United States
Jolly, Douglas J., Leucadia, CA, United States
PA Chiron Corporation, United States (U.S. corporation)
PI US 5698446 19971216
AI US 1994-305699 19940907 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Guzo, David
LREP Kruse, Norman J., Blackburn, Robert P.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2090
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for inhibiting the production of replication competent virus. The invention comprises nucleic acid cassettes encoding a non-biologically active inhibitory molecule which are incorporated into packaging cells and recombinant vector constructs. Upon recombination between various vector construct contained within the producer cell, a biologically active molecule is produced which kills the cell, thereby inhibiting production of replication competent virus.

L19 ANSWER 164 OF 178 USPATFULL
AN 97:80900 USPATFULL
TI **IL-12** inhibition of B1 cell activity
IN Metzger, Dennis W., Sylvania, OH, United States
Van Cleave, Victor H., Londonderry, NH, United States
PA Genetics Institute, Cambridge, MA, United States (U.S. corporation)
Medical College of Ohio, Toledo, OH, United States (U.S. corporation)
PI US 5665347 19970909
AI US 1995-382658 19950202 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Achutamurthy, Ponnathapura
LREP Hamilton, Brook, Smith & Reynolds, P.C.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1,2
DRWN 47 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 942
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to a method of suppressing B1 cell activity in a host (e.g., mammalian, including human) comprising administering to the

host an effective amount of **IL-12** that significantly suppresses or inhibits B1 cell activity. In addition, the invention relates to a method of treating a B1 cell disorder in a host, comprising administering to the host an effective therapeutic amount of **IL-12**. The invention further encompasses a method of screening for substances (e.g., proteins, peptides, small molecules) which enhance or suppress the inhibition of B1 cell activity by **IL-12**. The invention also relates to a substance identified by the methods of screening for a substance which enhances or suppresses **IL-12** inhibition of B1 cell activity.

L19 ANSWER 165 OF 178 USPATFULL
AN 97:68346 USPATFULL
TI Secreted proteins and polynucleotides encoding them
IN Jacobs, Kenneth, Newton, MA, United States
McCoy, John M., Reading, MA, United States
LaVallie, Edward R., Tewksbury, MA, United States
Racie, Lisa A., Acton, MA, United States
Merberg, David, Acton, MA, United States
Treacy, Maurice, Chestnut Hill, MA, United States
Spaulding, Vikki, Billerica, MA, United States
PA Genetics Institute, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5654173 19970805
AI US 1996-702080 19960823 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jagannathan, Vasu S.; Assistant Examiner: Lathrop, Brian
LREP Brown, Scott A., DesRosier, Thomas J.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1685
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polynucleotides and the proteins encoded thereby are disclosed.

L19 ANSWER 166 OF 178 USPATFULL
AN 97:64091 USPATFULL
TI P-40 homodimer of interleukin-12
IN Gately, Maurice Kent, Pine Brook, NJ, United States
Hakimi, John, Scarsdale, NY, United States
Ling, Ping, Nutley, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5650492 19970722
AI US 1995-424682 19950418 (8)
RLI Continuation of Ser. No. US 1993-87832, filed on 2 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John; Assistant Examiner: Mertz, Prema

LREP Johnston, George W., Tramaloni, Dennis P., Kass, Alan P.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analysis of the culture media of p40-transfected COS cells indicated the presence of 40 kDa monomers and 80 kDa disulfide-linked homodimers. Examination of partially purified p40 recombinant proteins demonstrated that only the homodimer but not the monomer binds to the **IL-12** receptor. Partially purified 80 kDa homodimer inhibited [.sup.125 I]**IL-12** binding to PHA-activated human lymphoblasts with an IC.sub.50 of 80 ng/ml, which is similar to the IC.sub.50 value (20 ng/ml) for the human **IL-12** heterodimer. Although neither the 40 kDa monomer nor the 80 kDa dimer could stimulate human PHA-blast proliferation, the 80 kDa dimer inhibited **IL-12**-induced proliferation in a dose-dependent manner with an IC.sub.50 of 1 .mu.g/ml. The **IL-12** p40 subunit contains the essential epitopes for receptor binding, but they are only active when p40 is covalently associated with a second protein such as p35 or p40. When p40 is associated with the p35 subunit, the heterodimer acts as an agonist mediating biologic activity. When p40 associates with itself, the homodimer behaves as an **antagonist**.

L19 ANSWER 167 OF 178 USPATFULL
AN 97:54233 USPATFULL
TI Substituted amino alcohol compounds
IN Klein, J. Peter, Vashon, WA, United States
Underiner, Gail E., Brier, WA, United States
Kumar, Anil M., Seattle, WA, United States
PA Cell Therapeutics, Inc., Seattle, WA, United States (U.S. corporation)
PI US 5641783 19970624
AI US 1994-303842 19940908 (8)
RLI Continuation-in-part of Ser. No. US 1993-152650, filed on 12 Nov 1993 And Ser. No. US 1993-164081, filed on 8 Dec 1993, now patented, Pat. No. US 5470878
DT Utility
FS Granted
EXNAM Primary Examiner: Raymond, Richard L.; Assistant Examiner: Cebulak, Mary C.
LREP Faciszewski, Stephen, Oster, Jeffrey B.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 115 Drawing Figure(s); 88 Drawing Page(s)
LN.CNT 3206
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are compounds having a straight or branched aliphatic hydrocarbon structure of formula I: ##STR1## In formula I, n is an integer from one to four and m is an integer from four to twenty. Independently, R.sub.1 and R.sub.2 are hydrogen, a straight or branched chain alkyl, alkenyl or alkynyl of up to twenty carbon atoms in length or --(CH.sub.2).sub.w R.sub.5. If R.sub.1 or R.sub.2 is --(CH.sub.2).sub.w R.sub.5, w may be an integer from one to twenty and R.sub.5 may be an hydroxyl, halo, C.sub.1-8 alkoxyl group or a substituted or unsubstituted carbocycle or heterocycle. Alternatively, R.sub.1 and R.sub.2 may jointly form a substituted or unsubstituted, saturated or unsaturated heterocycle having from four to eight carbon atoms, N being a hetero atom of the resulting heterocycle. R.sub.3 may be either hydrogen or C.sub.1-3. In the compounds, a total sum of carbon atoms comprising R.sub.1 or R.sub.2, (CH.sub.2).sub.n and (CH.sub.2).sub.m does not exceed forty. R.sub.4 is a terminal moiety comprising a substituted or unsubstituted, oxidized or reduced ring system, the ring system having a single ring or two to three fused rings, a ring comprising from three to seven ring atoms. The disclosed compounds are effective agents to inhibit undesirable responses to cell stimuli.

L19 ANSWER 168 OF 178 USPATFULL
AN 97:3820 USPATFULL
TI Genetic immunization
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
PI US 5593972 19970114
AI US 1993-125012 19930921 (8)
RLI Continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-8342, filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Fleisher, Mindy; Assistant Examiner: Railey, II, Johnny F.

LREP Woodcock Washburn Kurtz Mackiewicz & Norris
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 3611
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of prophylactic and therapeutic immunization of an individual
against pathogen infection, diseases associated with hyperproliferative
cells and autoimmune diseases are disclosed. The methods comprise the
steps of administering to cells of an individual, a nucleic acid
molecule that comprises a nucleotide sequence that encodes a protein
which comprises at least one epitope that is identical or substantially
similar to an epitope of a pathogen antigen, a hyperproliferative cell
associated protein or a protein associated with autoimmune disease
respectively. In each case, nucleotide sequence is operably linked to
regulatory sequences to enable expression in the cells. The nucleic acid
molecule is free of viral particles and capable of being expressed in
said cells. The cells may be contacted cells with a cell stimulating
agent. Methods of prophylactically and therapeutically immunizing an
individual against HIV are disclosed. Pharmaceutical compositions and
kits for practicing methods of the present invention are disclosed.

L19 ANSWER 169 OF 178 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993,
now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor

cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L19 ANSWER 170 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-648241 [74] WPIDS
DNC C2001-191222
TI N-Aryl 4-(optionally fused heteroaryl)-2-thiazolamines are **TNF** and IL cytokine inhibitors, useful for inflammatory and autoimmune disorders, e.g. **arthritis**, irritable bowel, transplants, asthma and shock.
DC B02 B03
IN COOYMANS, L; DE BRABANDER, M; KENNIS, L E J; LOVE, C; VAN WAUWE, J P F; VANDERMAESEN, N
PA (JANC) JANSSEN PHARM NV
CYC 94
PI WO 2001064674 A1 20010907 (200174)* EN 99p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001037401 A 20010912 (200204)
ADT WO 2001064674 A1 WO 2001-EP1841 20010220; AU 2001037401 A AU 2001-37401 20010220
FDT AU 2001037401 A Based on WO 200164674
PRAI EP 2000-200733 20000301
AB WO 200164674 A UPAB: 20011217
NOVELTY - Use of N-aryl 4-(optionally fused heteroaryl)-2-thiazolamines (I), or their N-oxides, simple and quaternary salts, and stereoisomers, for **treatment** and prophylaxis of cytokine mediated diseases.
DETAILED DESCRIPTION - Use of N-aryl 4-(optionally fused heteroaryl)-2-thiazolamines of formula (I), or their N-oxides, simple and quaternary salts, and stereoisomers, for **treatment** and prophylaxis of cytokine mediated diseases, is new.
Q = 3-6C cycloalkyl, phenyl, naphthyl, pyridyl, pyrimidinyl, pyrazinyl, pyridazinyl, benzothiazolyl, benzoxazolyl, benzimidazolyl, indazolyl, or imidazolyl (all optionally substituted by 1-3 J), or a hetero-fused phenyl group (a), (b), or (c):
J = halogen, hydroxy, cyano, carboxy, azido, amino, mono- or di-(1-6C alkyl)amino, 1-6C alkyl (optionally substituted), alkoxy, or

alkylthio, 2-6C alkenyl or alkynyl, 2-7C alkylcarbonyl or alkoxycarbonyl,
aryloxy, aryl 1-6C alkoxy, 1-4C alkylsulfinyl or alkylsulfonyl, or 1-4C
alkylaminosulfinyl, alkylaminosulfonyl or R1HN-S(=O)n-;
n = 0, 1 or 2;
X, Y = O, NR3, CH2, or S;
Z' = O or NR4;
q = 1-4;
r = 1-3;
L = phenyl or Het (both optionally substituted by 1-4 G, or 1-6 G for
fused bicyclic Het);
G = halogen, hydroxy, amino, cyano, carboxy, mono- or di-(1-6C
alkyl)amino, 1-6C alkyl (optionally substituted) or alkoxy, or 2-7C
alkylcarbonyl, alkylcarbonyloxy, alkylcarbonylamino, or
alkoxycarbonylamino, aminocarbonyl, or mono- or di- (1-6C
alkyl)aminocarbonyl;
aryl = phenyl (optionally substituted by 1-5 of halo, hydroxy,
(polyhalo) 1-6C alkyl, 1-6C alkyloxy, 1-6C alkylthio, cyano, nitro, amino
or mono- or di-(1-6C alkyl)amino);
R1 = H, or an azacyclic group (d):
R2a = H, or 1-6C alkyl or alkoxy;
A = O, S, or CR2a=N with the C attached to the NH; and
Het = 5 or 6 membered heterocyclyl containing 1-4 heteroatoms from N,
O, S and at least 2 double bonds, optionally fused through C or N atoms to
a 5 or 6 membered saturated, partially unsaturated, or aromatic, otherwise
carbocyclic or heterocyclic ring.

INDEPENDENT CLAIMS are also included for:

(1) the compounds of formula (I) with provisos. The provisos are
listed in FULL DEFINITIONS in the DEFINITIONS FIELD;

(2) several preparations of compound (I); and

(3) a composition comprising a new compound of formula (I) and
another antiinflammatory or immunosuppressive compound.

ACTIVITY - Antiinflammatory; antiarthritic; antiallergic; antiparasitic; antimalarial; antidiabetic; antiasthmatic; immunosuppressive; hepatotropic; nephrotrophic; vasotropic; tuberculostatic; vulnerary; antiparkinsonian; antithyroid; immunomodulator; antiviral; antirheumatic; dermatological; ophthalmological; antibacterial; antiparasitic; antipsoriatic; antiparkinsonian; antipyretic.

MECHANISM OF ACTION - (I) are inhibitors and/or **antagonists** of proinflammatory cytokines, notably **TNF**-alpha and/or **IL-12**. They also have selective affinity for, and block, the adenosine A3 receptor. Tests were conducted with cell free human
peripheral blood to determine inhibition of **TNF**- alpha and **IL-12** by compounds (I) at a concentration of 100 nM.

Respective results for a range of compounds were 39-56%, and 53-75% with

one 86%.

USE - For **treatment** or prevention of diseases mediated through activation of the adenosine A3 receptor (claimed). For use in the prevention and **treatment** of inflammatory or autoimmune disorders (such as rheumatoid **arthritis**, Crohn's disease, irritable bowel disease and colitis) (claimed). For **treatment** or prevention of diseases mediated through cytokines (specifically Tumor Necrosis Factor-alpha (**TNF**- alpha) and Interleukin 12 (**IL-12**) mediated diseases) (claimed).

For **treatment** of rheumatoid spondylitis, spondyloarthropathies, systemic lupus erythematosus, **arthritis**, polychondritis, sclerodoma, Wegener granulamatosis, dermatomyositis, Steven-Johnson syndrome, idiopatic sprue, endocrine opthalmopathy, Grave's disease, alveolitis, chronic hypersensitivity pneumonitis, primary billiary cirrhosis, uveitis, keratoconjunctivitis sicca and vernal keratoconjunctivitis, allergic rhinitis, pemphigus, eosinophilia, Loffler's syndrome, eosinophilic pneumonia, parasitic infestation, bronchopulmonary aspergillosis. polyarteritis nodosa, eosinophilic granuloma, eosinophil-related disorders affecting the airways occasioned by drug-reaction, sepsis, septic shock, endotoxic shock, gram negative sepsis, toxic shock syndrome, cerebral malaria, adult respiratory distress syndrome, bronchitis, chronic obstructive airway or pulmonary disease, pulmonary fibrosis, pneumocomosis, tuberculosis, silicosis, exacerbation of airways hyperreactivity to other drug **therapy** (e.g. aspirin or beta -agonist **therapy**), pulmonary sarcoidosis, bone resorption diseases, meningitis, reperfusion injury, graft versus host reaction, allograft rejections, transplant rejections, fever and royalgias due to infection, such as influenza, cachexia, AIDS, ARC (AIDS related complex), diabetes, cancer, angiogenesis, lymphoma, Kawasaki syndrome, Behcet's syndrome, aphthous ulceration, skin-related disorders (such as psoriasis and eczema), bowel disease (such as Crohn's disease), pyresis, asthma, wheezy infant syndrome, multiple **sclerosis**, Parkinson's disease, pancreatitis, cardiac disease, congestive heart failure, myocardial infarction, acute liver failure, glomerulonephritis, **therapy**-associated syndromes comprising Jarisch-Herxheimer reaction, and syndromes associated with IL-2 infusion, anti-CD3 **antibody** infusion, hemodialysis and yellow fever vaccination.

ADVANTAGE - (I) are stated to be more specific and less toxic than

present antiinflammatory and immunosuppressive drugs, and may be used in
combination with them to reduce dosing and side effects.
Dwg.0/0

L19 ANSWER 171 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-244697 [25] WPIDS
DNC C2001-073427
TI Modulating responsiveness to a corticosteroid by administering a corticosteroid with an agent which antagonizes a target that regulates interferon-gamma production or an caspase family protease inhibitor, useful for treating asthma.
DC B04 B05 D16
IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E
PA (BADI) BASF AG
CYC 94
PI WO 2001019373 A2 20010322 (200125)* EN 152p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000071276 A 20010417 (200140)
ADT WO 2001019373 A2 WO 2000-US24725 20000908; AU 2000071276 A AU 2000-71276
20000908
FDT AU 2000071276 A Based on WO 200119373
PRAI US 1999-398555 19990917
AB WO 200119373 A UPAB: 20010508
NOVELTY - A new method (M1) for modulating responsiveness to a corticosteroid in a subject comprises administering a corticosteroid with an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) or at least one agent (A2) that is an inhibitor of a caspase family protease.
DETAILED DESCRIPTION - A method (M1) for modulating responsiveness to a corticosteroid in a subject, comprising selecting a subject in need of modulation of responsiveness to a corticosteroid and administering:
(a) an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) in the subject, the agent being administered at a dosage and by a route sufficient to inhibit production of IFN-gamma; or
(b) at least one agent (A2) that is an inhibitor of a caspase family

protease; and

(c) a corticosteroid.

The responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

An INDEPENDENT CLAIM is also given for a method (M2) for regulating the production of IFN-gamma in a subject, comprising administering a corticosteroid and an agent which antagonizes a target that regulates production of IFN-gamma such that production of IFN-gamma is modulated in the subject.

ACTIVITY - Immunosuppressive; antiinflammatory; dermatological; antibacterial; cytostatic; antiasthmatic; anticonvulsant; antidiabetic; antiarthritic; antirheumatic; neuroprotective; antiallergic; antiulcer; ophthalmological; antianemic.

Interleukin converting enzyme (ICE)-deficient and wild type mice first were sensitized with Propionibacterium acnes cell wall material (1 mg per mouse) to induce low grade inflammation and six days later were challenged with lipopolysaccharide (LPS) (1 microgram per mouse in 0.1 ml of saline intravenously). Thirty minutes after LPS administration, the mice were treated with the corticosteroid dexamethasone (4 mg/kg per mouse in 0.5 ml 95% saline/0.5% ethanol, intraperitoneally). Control mice were treated with vehicle alone. All mice were bled 90 minutes after LPS administration and the serum samples were analyzed for the presence of tumor necrosis alpha (**TNF**-alpha) by standard ELISA (Enzyme linked immunosorbant assay).

Wild type and ICE deficient mice treated with vehicle alone had similar levels of serum **TNF**-alpha. **Treatment** of wild type mice with dexamethasone did not significantly affect serum **TNF**-alpha levels, demonstrating their resistance to steroid **treatment** in this septic shock model. In contrast, **treatment** of the ICE deficient mice with dexamethasone suppressed serum **TNF**-alpha levels by 74% (p less than 0.002). These data indicate that inhibition of ICE activity reverses resistance to steroid **treatment** in a septic shock model.

MECHANISM OF ACTION - **IL-12 antagonist**; IL-18 **antagonist**; phosphodiesterase IV inhibitor; a beta-2 agonist; a STAT4 inhibitor; an anti-IL-1-alpha **antibody**; an anti-IL-1-beta **antibody**; an anti-tumor necrosis factor **antibody**; a natural killer cell **antagonist**; a T-cell **antagonist**; caspase family protease inhibitor; gene

therapy.

USE - The method is useful for treating a subject suffering from an autoimmune disease or disorder, an acute (e.g. infectious meningitis) or chronic (e.g. systemic lupus erythematosus or psoriasis) inflammatory disorder, septic shock or sepsis, graft versus host disease or transplant rejection, complications associated with post-surgical stress, Still's disease, leukemia or an immuno-inflammatory disease or disorder. The immuno-inflammatory disease or disorder is asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple **sclerosis**, insulin-dependent diabetes mellitus, autoimmune **arthritis**, rheumatoid **arthritis**, juvenile rheumatoid **arthritis**, psoriatic **arthritis**, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior or interstitial lung fibrosis (claimed).

The method is useful for modulating corticosteroid responsiveness in a variety of clinical settings, for e.g. reversing steroid resistance, increasing steroid sensitivity, ameliorating a steroid rebound effect associated with administration of reduced dosages of the corticosteroid, or modulating corticosteroid activity, such that the corticosteroids can be tapered to zero (claimed).

Dwg.0/12

L19 ANSWER 172 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD

AN 2001-244560 [25] WPIDS

DNC C2001-073385
TI Composition comprising interleukin-12 p40 and IL-B30 polypeptide or its segment, useful for ameliorating rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple **sclerosis**, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 91
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ
EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV
MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT
TZ UA UZ VN YU ZA
AU 2000073608 A 20010410 (200137)
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608
20000908
FDT AU 2000073608 A Based on WO 200118051
PRAI US 1999-164616P 19991110; US 1999-393090 19990909
AB WO 200118051 A UPAB: 20010508
NOVELTY - A composition (I) comprising a substantially pure polypeptide
comprising a number of distinct segments of at least 7 contiguous amino
acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and
a substantially pure polypeptide comprising a segment of at least 11
contiguous amino acids from **IL-12** p40 and/or IL-B30.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:
(1) an isolated or recombinant nucleic acid (II) encoding (I);
(2) a cell (III) comprising (II);
(3) a nucleic acid (IV) which hybridizes under wash conditions of 30
minutes at 50 deg. C and less than 1M salt to the natural mature coding
portion of primate **IL-12** p40 and IL-B30;
(4) an **antagonist** (V) of **IL-12**
p40/IL-B30 combined with a tumor necrosis factor-alpha (**TNF** alpha) **antagonist**, an **IL-12**
antagonist, IL-10, or steroids;
(5) a binding compound (VI) comprising an antigen binding site from
an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12** p40
and IL-B30 polypeptide, or a polypeptide comprising **IL-12** p40 fused to IL-B30, but not to either **IL-12**

p40 or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12** p40 or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12** p40/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves expressing the polypeptide with **IL-12** p40 or increasing the secretion of a primate **IL-12** p40 involves expressing the **IL-12** p40 with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, **IL-12**, radiation **therapy** or chemotherapy, an immune adjuvant or an anti-viral therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The

antagonist or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steriod. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple **sclerosis**, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

IL-12 p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L19 ANSWER 173 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2000-422868 [36] WPIDS
CR 1996-268530 [27]; 1998-377241 [29]; 2000-061893 [05]; 2000-071668 [05]; 2000-170770 [05]
DNC C2000-127890
TI Therapeutic **treatment** of for example viral diseases such as chronic hepatitis B and C, cancers such as leukemia, and multiple **sclerosis** comprises administering an immunological tolerance inducing compound prior to an effective drug .
DC B04 D16
IN TOVEY, M G
PA (PHAR-N) PHARMA PACIFIC PTY LTD
CYC 21
PI WO 2000032223 A2 20000608 (200036)* EN 26p
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU JP US
AU 2000013991 A 20000619 (200044)
ADT WO 2000032223 A2 WO 1999-GB4009 19991201; AU 2000013991 A AU 2000-13991

19991201
FDT AU 2000013991 A Based on WO 200032223
PRAI EP 1998-403020 19981202
AB WO 200032223 A UPAB: 20000801
NOVELTY - Therapeutic **treatment** of a subject with an immunogenic drug comprising:
(a) administering oromucosally a first formulation comprising a compound which induces immunological tolerance to the drug; and
(b) administering a second formulation comprising the drug that effects the therapeutic **treatment**.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) A kit for therapeutic **treatment** of a subject with an immunogenic drug comprising a formulation comprising a compound to induce immunological tolerance to the drug and a formulation comprising the drug to effect the therapeutic **treatment**;
(2) Using an immunogenic drug for the manufacture of a formulation to effect therapeutic **treatment** of a disease of a human or animal which has become immunologically tolerant to the drug by the oromucosal route of a formulation comprising a compound that induces immunological tolerance; and
(3) Using a compound for the manufacture of a formulation for oromucosal administration to a human or animal to induce immunological tolerance to an immunological drug where the human or animal is also administered a second formulation comprising the drug to effect a therapeutic effect.
ACTIVITY - Virucide; Cytostatic; Neuroprotective; Immunostimulant; Antianemic; Antibacterial; Immunosuppressive; Antirheumatic; Antiarthritic.
MECHANISM OF ACTION - None given.
USE - For therapeutic **treatment** of a human or animal. An immunogenic drug or compound is used to manufacture formulations for inducing an immunological tolerance or effecting therapeutic **treatment** (claimed). Viral diseases, such as chronic hepatitis B and C, herpes, and influenza; cancers, such as leukemia, lymphomas and solid tumors; and multiple **sclerosis** are treated. Neutropenia and leukopenia following chemotherapy are treated. Anemia, chronic renal failure. septic shock and rheumatoid **arthritis** are treated. Cystic fibrosis and Gaucher disease can be treated by gene **therapy**.
ADVANTAGE - An immunological tolerance to an immunogenic drug is induced so that when the drug is subsequently administered, its

pharmacokinetics and/or clinical effectiveness are improved. Rejection of drugs that are administered in repeat doses over a period of time by the immune system is less likely. The amount of drug that needs to be administered is reduced, lowering costs. Non-humanized **antibodies** that cannot normally be used for **therapy** due to rejection by the immune system can be used.
Dwg.0/0

L19 ANSWER 174 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2000-182039 [16] WPIDS
DNN N2000-134380 DNC C2000-056809
TI A process for expanding and selecting disease associated T-cells useful for the production of vaccines.
DC B04 D16 S03
IN ANGHOLT, J; KALTOFT, K; AGNHOLT, J
PA (AGNH-I) AGNHOLT J; (KALT-I) KALTOFT K; (CELL-N) CELLCURE APS
CYC 87
PI WO 2000000587 A1 20000106 (200016)* EN 124p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB
GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU
LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR
TT UA UG US UZ VN YU ZA ZW
AU 9946034 A 20000117 (200026)
EP 1090104 A1 20010411 (200121) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
ADT WO 2000000587 A1 WO 1999-DK363 19990625; AU 9946034 A AU 1999-46034 19990625; EP 1090104 A1 EP 1999-929110 19990625, WO 1999-DK363 19990625
FDT AU 9946034 A Based on WO 200000587; EP 1090104 A1 Based on WO 200000587
PRAI US 1998-91684P 19980702; DK 1998-848 19980626; DK 1998-895 19980701
AB WO 200000587 A UPAB: 20000330
NOVELTY - A method (A) of expanding and selecting disease associated T-cells comprises: (a1) obtaining a tissue sample from a mammal including a human being, comprising disease activated T-cells, or (a2) obtaining T-cells and antigen-presenting cell from the mammal and mixing the cells with a disease associated antigen or antigens; and (b) culturing the tissue sample or the mixture of cells and antigen(s) in the presence of at least 2 factors which promote T-cell growth and optionally at least 1

additional compound.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a vaccine comprising activated disease associated inflammatory T-cells prepared by (A);

(2) a pharmaceutical composition for use in an adjuvant **treatment** of a disease comprising disease associated regulatory or cytotoxic T-cells prepared by (A);

(3) a method for the diagnosis of a disease in a mammal, comprising:

(a) obtaining a tissue sample from a mammal including a human being, the sample comprising activated T-cells, antigen presenting cells and antigen(s); and

(b) culturing the tissue sample or the activated T-cells in the presence of two or more T-cell growth factors and optionally one or more additional compound; a method for the **treatment**, alleviation or prevention of a disease associated with an activation of T-cells in a subject comprising administering a T-cell line produced as described above;

(4) a model system for testing the effect of a medicament against a T-cell associated disease comprising at least one T-cell line as described above;

(5) a method for the **treatment**, alleviation or prevention of a disease associated with an activation of T-cells in a subject comprising administering (2); and

(6) a method of monitoring the response to a **treatment** of a disease of inflammatory, auto-immune, allergic, neoplastic or transplantation-related origin, or combinations thereof, comprising comparing the phenotype proliferation, apoptosis, cytokine profile, intracellular amount of NFKB and/or JAK/STAT pathway of activated Tcells in tissue sample taken from the patient to be treated before the start of the **treatment** and during the **treatment** and/or after the **treatment** has ended.

USE - The disease associated T-cells are associated with a disease of inflammatory, auto-immune, allergic, neoplastic and/or transplantation-related origin. The disease of inflammatory or allergic origin is a chronic inflammatory disease, or a chronic allergic disease.

The disease is an chronic inflammatory bowel disease, such as Crohn's disease or ulcerative colitis, **sclerosis**, type I diabetes, rheumatoid **arthritis**, psoriasis, atopic dermatitis, asthma,

malignant melanoma, renal carcinoma, breast cancer, lung cancer, cancer of
the uterus, prostatic cancer, cutaneous lymphoma, hepatic carcinoma,
rejection-related disease, or Graft-versus-host-related disease.
Dwg.0/22

L19 ANSWER 175 OF 178 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 1998-261495 [23] WPIDS
DNC C1998-081292
TI New compositions for immuno-**therapy** and protection - comprise
nucleotide sequences encoding an immuno-modulating protein and an antigen,
used for e.g. infections, cancer or auto-immune diseases.
DC B04 C06 D16
IN BAGARAZZI, M L; BOYER, J D; KIM, J J; WANG, B; WEINER, D B; AYYAVOO, V
PA (APOL-N) APOLLON INC; (UYPE-N) UNIV PENNSYLVANIA
CYC 80
PI WO 9817799 A1 19980430 (199823)* EN 136p
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT
SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE
GH HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN
MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ
VN YU ZW
AU 9750022 A 19980515 (199838)
EP 958364 A1 19991124 (199954) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
BR 9712852 A 19991116 (200012)
CN 1242045 A 20000119 (200023)
AU 729579 B 20010201 (200112)
KR 2000052710 A 20000825 (200121)
JP 2001507216 W 20010605 (200138) 141p
ADT WO 9817799 A1 WO 1997-US19502 19971023; AU 9750022 A AU 1997-50022
19971023; EP 958364 A1 EP 1997-912961 19971023, WO 1997-US19502 19971023;
BR 9712852 A BR 1997-12852 19971023, WO 1997-US19502 19971023; CN 1242045
A CN 1997-180897 19971023; AU 729579 B AU 1997-50022 19971023; KR
2000052710 A WO 1997-US19502 19971023, KR 1999-703507 19990422; JP
2001507216 W WO 1997-US19502 19971023, JP 1998-519714 19971023
FDT AU 9750022 A Based on WO 9817799; EP 958364 A1 Based on WO 9817799; BR
9712852 A Based on WO 9817799; AU 729579 B Previous Publ. AU 9750022,
Based on WO 9817799; KR 2000052710 A Based on WO 9817799; JP 2001507216 W
Based on WO 9817799
PRAI US 1996-28613P 19961023
AB WO 9817799 A UPAB: 19980610
The following are claimed: (A) A plasmid which comprises a nucleotide

sequence (NS) that encodes:(a) an immunomodulating protein selected from interleukin (**IL**)-**12**, granulocyte-macrophage colony stimulating factor (GM-CSF), IL-1, tumour necrosis factor (**TNF**)-alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and BL-1 operably linked to regulatory elements; (b) a NS that encodes an immunogen; (B) a composition comprising at least 2 plasmids including a first plasmid comprising a NS that encoded an immunomodulating protein selected from **IL-12**, GM-CSF, IL-1, **TNF**-alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and BL-1 operably linked to regulatory elements; and a second plasmid comprising a NS that encodes an immunogen; (C) a recombinant vaccine comprising a NS that encodes an immunomodulating protein selected from **IL-12**, GM-CSF, IL-1, **TNF**- alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18 and BL-1 operably linked to regulatory elements; and a second plasmid comprising a NS that encodes an immunogen; (D) a live attenuated pathogen comprising a NS that encodes an immunomodulating protein selected from **IL-12**, GM-CSF, IL-1, **TNF**- alpha , **TNF**- beta , IL-2, IL-4, IL-5, IL-10, IL-15, IL-18, and BL-1 operably linked to regulatory elements; (E) a plasmid comprising a NS that encodes single chain **IL-12**; (F) a pure BL-1 protein having an amino acid sequence given in the specification, or an immunomodulatory fragment; (G) a recombinant expression vector comprising a nucleic acid sequence that encodes a protein as in (F); (H) an isolated **antibody** which binds to an epitope on a protein as in (F).

The immunogen in (A) is a target protein operably linked to regulatory segments, where the target protein encodes a pathogen antigen, a cancer-associated antigen or an antigen linked to cells associated with autoimmune diseases. It is preferably an HIV-1 antigen. The immunomodulatory protein is a single chain **IL-12**. The **antibody** (H) is a monoclonal **antibody**.

USE - The products can be used to induce an immune response to an antigen such as a pathogen antigen, a hyperproliferative disease-associated antigen, and antigen linked to cells associated with autoimmune diseases or an allergen. They can be used for immunotherapy or to provide a protective immune response. In particular, they can be used for treating subjects with an allergic reaction, pathogen infection, hyperproliferative disease such as cancer or psoriasis or autoimmune

diseases e.g. rheumatoid **arthritis** (RA), multiple **sclerosis** (MS), Sjogren's syndrome, sarcoidosis, insulin dependent diabetes mellitus (IDDM), autoimmune thyroiditis, reactive **arthritis**, ankylosing spondylitis, scleroderma, polymyositis, dermatomyositis, psoriasis, vasculitis, Wegener's granulomatosis, Crohn's disease and ulcerative colitis, lupus (SLE), Grave's disease, myasthenia gravis, autoimmune haemolytic anaemia, autoimmune thrombocytopenia, asthma, cryoglobulinaemia, primary biliary **sclerosis** and pernicious anaemia.
Dwg.0/17

L19 ANSWER 176 OF 178 CAPLUS COPYRIGHT 2002 ACS
AN 2000:688272 CAPLUS
DN 133:280563
TI Human **antibodies** that bind human **IL-12** and methods for producing
IN Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PA Basf A.-G., Germany; Genetics Institute Inc.; et al.
SO PCT Int. Appl., 377 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000056772	A1	20000928	WO 2000-US7946	20000324

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-126603 P 19990325
AB Human **antibodies**, preferably recombinant human **antibodies**, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred **antibodies** have high affinity for hIL-12 and neutralize hIL-12 activity in vitro and in vivo . An

antibody of the invention can be a full-length **antibody** or an antigen-binding portion thereof. The **antibodies**, or **antibody** portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental.

Nucleic acids, vectors and host cells for expressing the recombinant human **antibodies** of the invention, and methods of synthesizing the recombinant human **antibodies**, are also encompassed by the invention.

RE.CNT 7
RE
(2) Carter, R; HYBRIDOMA 1997, V16(4), P363 CAPLUS
(3) Genentech Inc; WO 9404679 A 1994 CAPLUS
(4) Genetics Inst; WO 9524918 A 1995 CAPLUS
(5) Irving, R; IMMUNOTECHNOLOGY 1996, V2(2), P127 CAPLUS
(6) Pini, A; JOURNAL OF IMMUNOLOGICAL METHODS 1997, V206(1-2), P171 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 177 OF 178 CAPLUS COPYRIGHT 2002 ACS
AN 1995:934127 CAPLUS
DN 123:337469
TI Use of **IL-12** and **IL-12** **antagonists** in **treatment** of autoimmune diseases
IN Leonard, John P.; Goldman, Samuel; O'Hara, Richard, Jr.
PA Genetics Institute, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09510444	T2	19971021	JP 1995-524044	19950307
	US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		

AB Autoimmune conditions such as multiple **sclerosis**, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye

disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or **TNF**-.alpha., are treated in mammals by administering **IL-12** or an **IL-12 antagonist**. Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with **IL-12** during restimulation, and alleviated by injection of a polyclonal **antibody** to **IL-12**.

L19 ANSWER 178 OF 178 BIOTECHDS COPYRIGHT 2002 DERWENT INFORMATION LTD
AN 2001-08257 BIOTECHDS
TI Composition containing interleukin-12 p40 and IL-B30 protein or its segment, useful for ameliorating rheumatoid **arthritis**, osteoarthritis, atherosclerosis, multiple **sclerosis**, vasculitis and tumor;
vector-mediated gene transfer and expression in host cell, **antibody** and **antagonist**
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K
PA Schering-USA
LO Kenilworth, NJ, USA.
PI WO 2001018051 15 Mar 2001
AI WO 2000-US24686 8 Sep 2000
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999
DT Patent
LA English
OS WPI: 2001-244560 [25]
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous amino acids from **IL-12** p40 and/or IL-B30, is new. Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural mature coding portion of primate **IL-12** p40 and IL-B30; an **antagonist** of **IL-12** p40/IL-B30 combined with a tumor necrosis factor-alpha (**TNF**-alpha) **antagonist**, an **IL-12 antagonist**, IL-10 or steroids; a binding compound containing an antigen binding site from an **antibody** which specifically binds to the protein; a kit containing the composition, polynucleotide and a binding compound; producing an antigen:**antibody** complex; a composition containing

a binding compound; increasing the secretion of a primate IL-B30; and

screening for a receptor which binds the composition. The composition is

useful for modulating physiology or development of a cell or tissue0.

(69pp)